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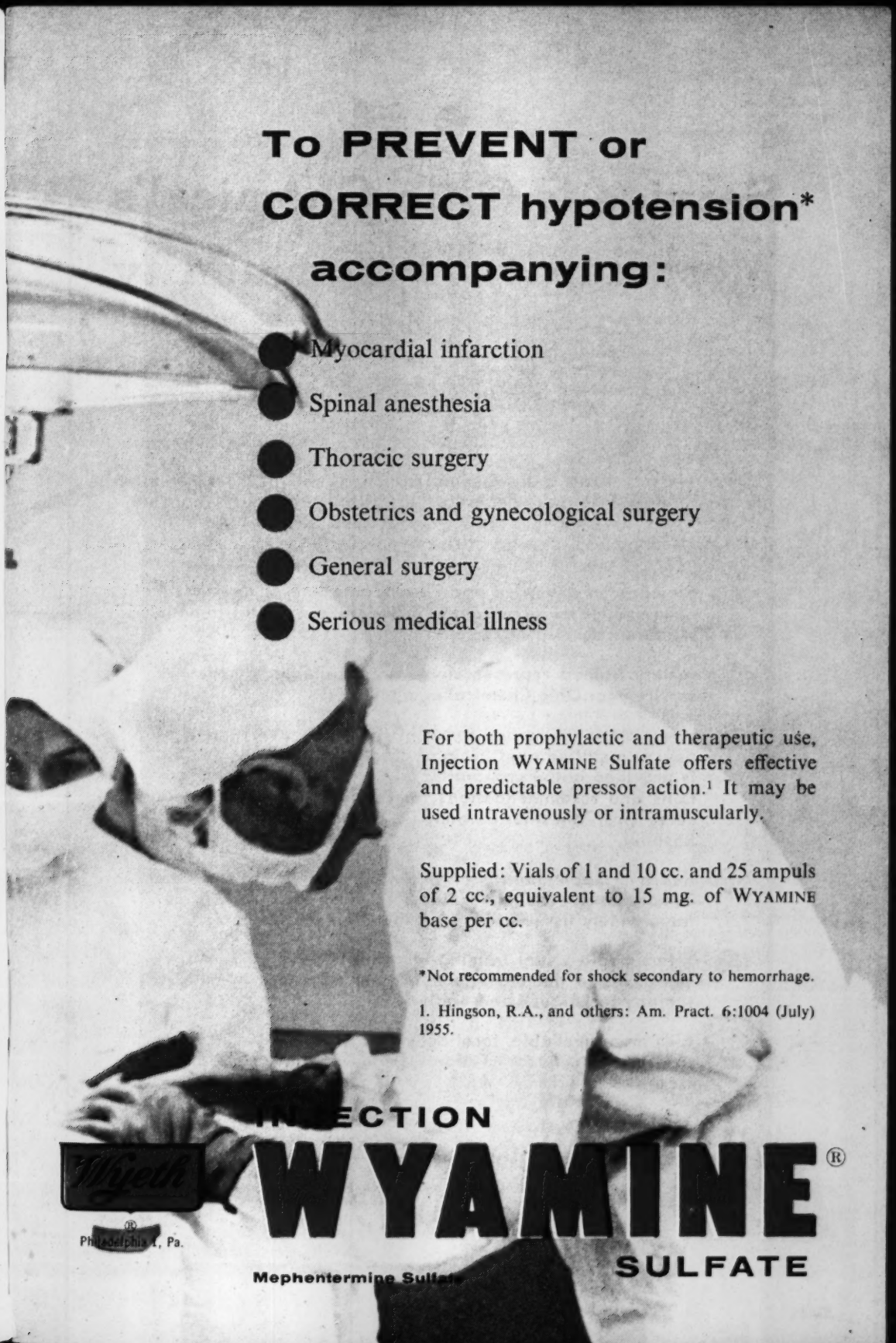

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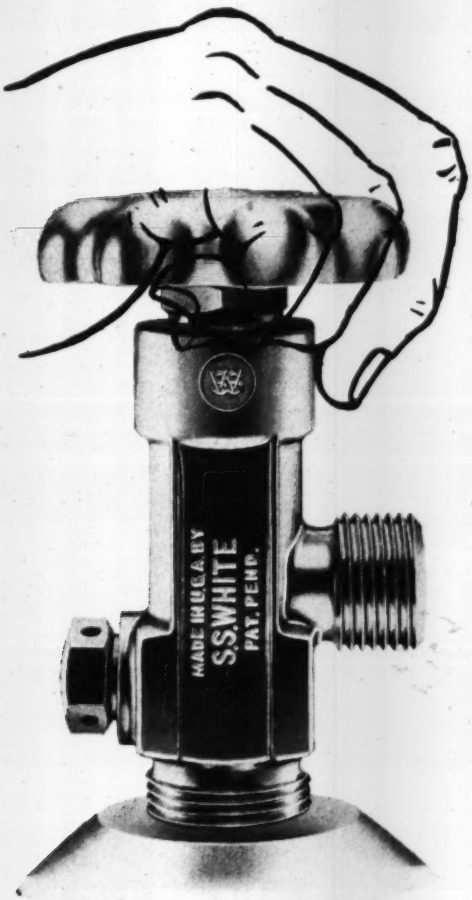

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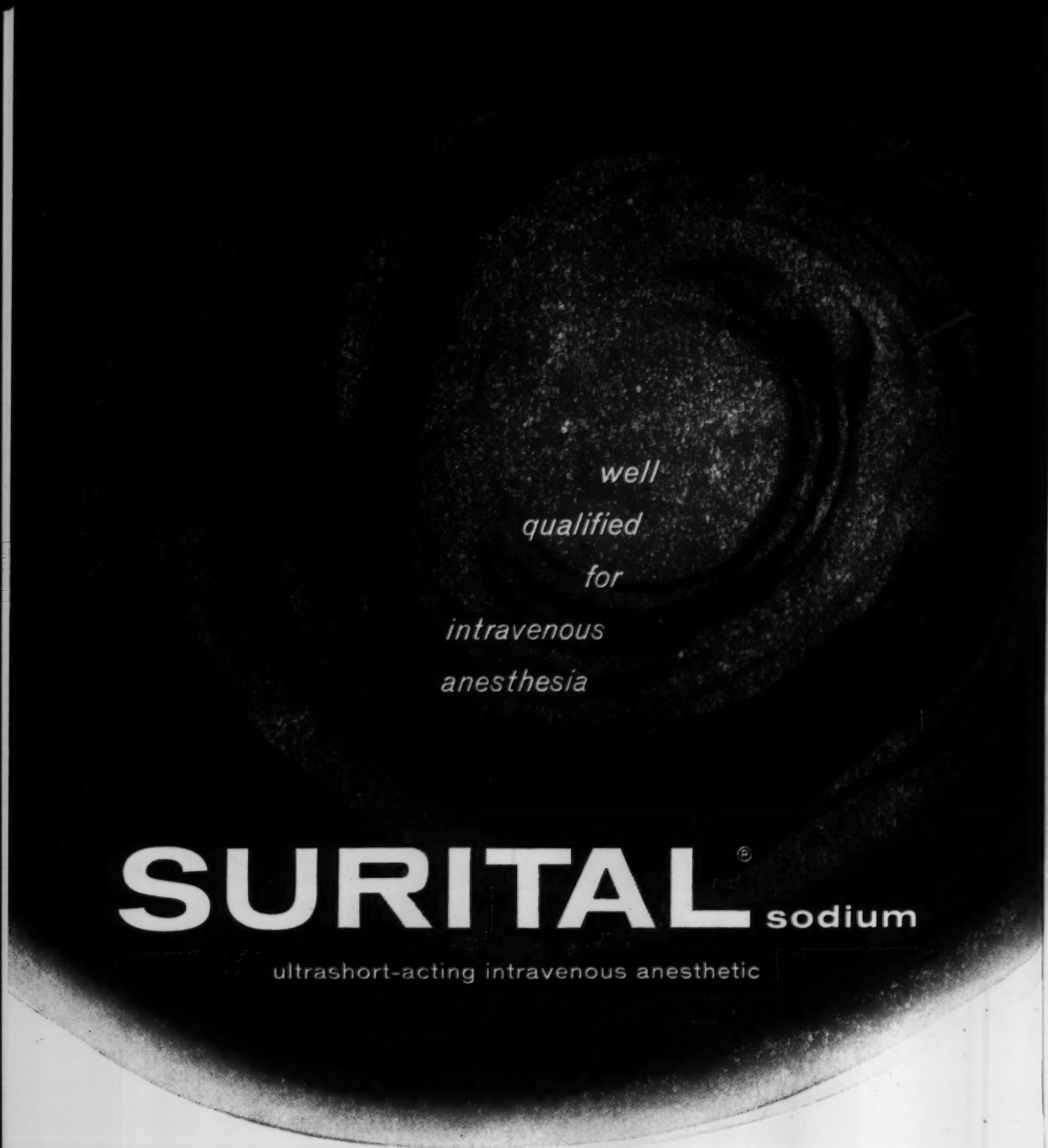

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Vomiting and Regurgitation During and After Anesthesia

Some Causes, Effects, Prevention and Management

John Adriani, M.D.*
New Orleans, Louisiana

Few happenings are as disconcerting to an anesthetist as persistent postoperative emesis particularly if the patient underwent anesthesia uneventfully. Since the day ether was first used postoperative emesis has been associated with anesthesia both in the minds of professional personnel who care for the sick and the public. No doubt anesthesia plays a role in this vexsome complication but it has been awarded more than its share of the blame.

The problem resolves itself into two phases: that of emesis during anesthesia and that of emesis in the postanesthetic period. Both aspects of the problem are important but emesis during anesthesia is a more serious situation than emesis after. The majority of fatalities on the operating table are due to asphyxia. Aspiration of vomitus, blood and other secretions account for more than half the asphyxial deaths. Emesis during anesthesia can be averted. When it occurs the anesthetist invariably is at fault. Lack of skill, improper preparation or failure to properly evaluate a patient preoperatively are the factors involved.

A knowledge of the physiology involved in emesis is important. The terms vomiting and regurgitation are used interchangeably at times. There is a difference between them and this difference is important, particularly in prophylaxis. Vomiting is an active response in which some voluntary effort is involved. Regurgitation is passive and involves no voluntary effort. The impulses which initiate vomiting arise in neurons in the vomiting center which is located in the medulla. Vomiting cannot occur unless the center is active. Anesthetics depress the center. Emesis during general anesthesia occurs because the patient becomes "light" and passes from third into the second or first stage. Emesis is accomplished by the coordinated contraction of visceral smooth muscle and certain groups of striated muscle. The sequence of events is as follows: the abdominal muscles and the diaphragm, which is also striated muscle, contract simultaneously and compress the stomach. A wave of reverse peristalsis is initiated in the stomach and esophagus so that the gastric contents are propelled into the pharynx. As the vomitus reaches the upper esophagus the muscles of the pharynx, those at

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the base of the tongue, and those of the tongue itself come into play in such a manner that the vomitus is propelled from the hypopharynx and into the mouth. All of these movements are coordinated by the vomiting center.

The existence of a vomiting center has been postulated for many years but its presence was only demonstrated recently. The fact that motor functions involved in emesis can be reproduced with striking regularity is presumptive evidence that a center coordinates the act since it requires complex integration. A network of cells extends from the medulla to the cerebrum called the reticular formation. Electrical stimulation of the reticular formation in the medulla causes emesis indicating that the center is located in this reticulum. By the technique of electrical mapping, which consists of stimulation of various areas with fine minute electrodes, it was possible to locate this center. If the center is destroyed by radiation the ability to vomit is abolished. The vomiting center lies close to other vital medullary centers, notably those subserving spasmodic respiration, salivation, inspiration, expiration, cardiovascular control and vestibular functions. Presumably there is some interrelation between these vital centers and the vomiting center. In addition to the vomiting center, which is often referred to as the true vomiting center, there is a chemoreceptor zone located in the floor of the fourth ventricle in the medulla close to the true vomiting center. This center is sensitive to chemical substances which induce vomiting. Substances such as copper sulphate and apomorphine act upon the cells in this center. Cells in this area do not respond to electrical stimuli. Chemical stimulation apparently is the only manner by which it is activated. Emetic agents such as apomorphine and copper sulphate produce no vomiting if injected after this center is destroyed. Chemical substances such as apomorphine and copper sulphate act only on the chemical receptor center and not on the true vomiting center. The chemical center is stimulated first by these drugs and this in turn stimulates the true vomiting center which initiates the act. Groups of neurons are also present above the medulla which influence the vomiting center and induce vomiting. Vomiting due to psychic influences and to neurologic lesions, such as brain tumors, suggest this. Definite evidence exists that emesis may result from some stimuli which are relayed downward from the cerebral cortex.

Impulses which initiate [redacted] may originate in almost a[redacted] the body because the vomit[redacted]er is in communication with many [redacted]rves from many areas. Impulses from the stomach and intestines reach the medulla by way of the visceral fibres of the vagus and the sympathetic trunks. Impulses are relayed to the center along somatic nerves such as the phrenic, the intercostals, the spinal nerves from the skin, muscles, bones, joints and other external structures. These impulses pass to the medulla. Vomiting has long been associated with parasympathetic activity. Vagal receptors are present in the pharynx and larynx which are stimulated and initiate vomiting and retching. Laryngoscopes and pharyngeal airways may initiate gagging, retching and vomiting. The vomiting center transmits impulses to the skin, salivary glands and the heart which are responsible for salivation, sweating and bradycardia which often accompany vomiting. Vomiting result-

ing from motion is indistinguishable from that caused by other stimulation. Impulses in motion sickness arise in the labyrinth. Destruction of the labyrinth prevents vomiting from this source. Vomiting, when patients are moved after they react, may originate in the labyrinth. Thus, one can see that stimuli from any area of the body may induce vomiting and that the reflex can spread to affect other organs. Therefore, in order to properly treat persistent emesis, one must know its cause and mechanism of action.

Many of the drugs used in anesthesia, particularly the narcotics and the general anesthetics, may stimulate the vomiting centers in the medulla. Various therapeutic agents may produce vomiting. The cardiac glucosides of the digitalis type may induce vomiting by action on the cardiac receptors. The veratrum alkaloids possibly act somewhat in the fore and midbrain and induce emesis. Morphine probably acts on the chemoreceptors instead of the true vomiting center. Anesthetics may behave similarly. Some stimuli arise in the periphery instead of centrally. Drugs may stimulate receptors in the intestinal mucosa where they are relayed to the medulla and induce vomiting. Some workers feel that the small amounts of ether which are secreted into the stomach during anesthesia may cause local stimulation which induces emesis. The anti-emetic drugs act principally on the chemical receptor center. Inasmuch as vomiting appears to be related to cholinergic activity it is not unreasonable to find that anticholinergic activity lessens the incidence of nausea and vomiting. For this reason atropine and scopolamine are frequently effective in overcoming vomiting. The anti-emetic drugs such as dramamine, marazine and chlorpromazine depress the chemoreceptor area also. It is possible that they may also act on the true vomiting center by depressing it but the exact mode of action has not been established as yet since these drugs are new.

Regurgitation not only occurs without voluntary effort but even when the vomiting center is depressed. Vomiting, on the other hand, does not occur if the vomiting center is depressed by anesthetics. Regurgitation is due to relaxation of the cardiac sphincter which makes possible the reflux of fluid from the stomach, particularly when the stomach and intestines are dilated and are filled with fluid. Regurgitation in these cases is usually massive, silent and occurs abruptly. Often it is initiated by manipulations of the stomach and intestines by the surgeon, particularly during upper abdominal surgery. It is far more frequent in cases in which there has been obstruction or stasis in the intestines and the stomach. Regurgitation may occur at any time during induction or as anesthesia deepens. It is more difficult for the anesthetist to avoid regurgitation than vomiting.

Vomiting during anesthesia may be subdivided into that which occurs during induction and that during maintenance. The most common causes of vomiting during induction are as follows:

1. Difficult or prolonged induction from improper premedication.
2. Incorrect use of agents which result in a prolonged stormy induction.

It is difficult to impress upon physicians in the surgical specialties that patients with a full stomach invariably attempt to evacuate it if anesthetized. This occurs irrespective of the type of anesthesia used, including local and spinal. Inasmuch as stimuli

which initiate vomiting arise in many portions of the body vomiting may be frequent in conscious patients undergoing surgery. It is possible for conscious patients to regurgitate, or vomit and aspirate and die of asphyxia because the supine position favors aspiration. The patient undergoing surgery is restrained and cannot lift his head to protect himself from aspiration. It is erroneous to assume that because the pharyngeal and laryngeal reflexes are active that a patient who is conscious cannot aspirate. Acutely ill or semi-comatose "toxic" patients have obtunded laryngeal and tracheal reflexes. They aspirate whether or not they are under the influence of a general anesthetic. The writer has seen patients drown from their own vomitus while undergoing surgery with local, spinal and intravenous anesthesia. No type of anesthesia assures immunity from this catastrophy.

Inasmuch as the full stomach accounts for the majority of difficulties it merits further discussion. The management of the patient with a full stomach has been a matter of debate for sometime. There is no unanimity among clinicians as to what procedure to follow. Some clinicians advocate emptying the stomach by lavage, using a large stomach tube. In the experience of the writer this is not a good practice because one is never sure that all the gastric contents have been evacuated. Seldom does one retrieve all of the washings which are introduced into the stomach. In a normal individual a full stomach normally is totally evacuated within three to four hours. The patient who is ill, in pain, injured, apprehensive or who has had medication, particularly narcotics, responds differently. Ingested food may remain undigested in the stomach for many hours. The writer has seen undigested food in stomachs 18 hours after the patient had partaken of a meal. Perhaps the wisest course to adopt in managing patients with full stomachs is to defer operation for a day when possible. No rule of the thumb can be adopted concerning the number of hours one must wait to be assured that the stomach is empty. There are too many variable factors involved, such as the make-up of the patient, drugs used for therapy or pain relief, the extent of the physical trauma, the presence of shock and so on. One has no assurance that the stomach will be completely evacuated within five, ten or even fifteen hours. The use of drugs such as apomorphine to induce vomiting is suggested from time to time. This is drastic treatment. Apomorphine is a central depressant. Respiratory and circulatory depression may develop later during anesthesia. Its use may jeopardize the patient who is acutely ill and many urgent operations are performed on acutely ill patients.

Aspiration of undigested food accounts for many maternal deaths. Food should be withheld from the patient in labor when one contemplates using general anesthesia for delivery. Obstetrical anesthesia for delivery differs in no way from surgical anesthesia for elective operations. The preparation and management should be identical. A fasting patient is required. One would be horrified at the thought of permitting a patient scheduled for an elective operation to eat breakfast. Still certain obstetricians nonchalantly allow patients in labor to have solid food.

When surgery is urgent and the operation must proceed, the best expedient is to effect a rapid induction with cyclopropane or pentothal with a muscle relaxant. Intubation of the patient using a cuffed tube is mandatory when vomiting is anticipated.

Thus, if during maintenance of anesthesia the patient inadvertently passes from stage III to stage II, in which case vomiting invariably occurs, the cuff will afford full protection. The anesthetist should be prepared to place the patient in a steep head down position at the conclusion of anesthesia. The angle of inclination should be as great as 45°. One must bear in mind that the trachea normally points posteriorily at angle of 10° or more when the patient is in the supine position. When the table is inclined approximately 15° the trachea is parallel to the floor. In other words, it is horizontal. Therefore, a 35 to 40° position is needed to have the trachea inclined 10 or 15°. A forceful suction apparatus equipped with a throat suction tip with large perforations should be on hand to remove solid particles of vomitus. Catheters are worthless in situations when massive regurgitation or vomiting occurs because they tend to kink and cannot be directed to all areas of the pharynx because of lack of rigidity. Besides, it is often difficult to insert a catheter between the teeth of patients who are regaining their reflexes. Often patients bite down on the catheter if they are "reacting". The endotracheal catheter should be left in position until the patient has fully recovered. Care should be exercised to fix the bite block with adhesive since it may be dislodged as the patient becomes active and he may bite down on the tracheal catheter during emergence. One will then be compelled to remove the catheter prematurely. The best place to manage vomiting is in the operating room. One should keep the patient there until danger from this complication has passed. It is far simpler to cope with vomiting where the suction and a tilting operating table are available than to attempt to cope with it en-route to the recovery room or in the recovery room itself. Certain writers have suggested that celluloid masks be used in order to visualize vomiting. One must remember that long before the vomitus appears at the lips enough of it may have passed into the trachea to be fatal. This suggestion is one which would not lessen the danger. It would do nothing more than to make the anesthetist complacent. Recently certain writers have suggested using stomach tubes with inflatable cuffs. This tube can be introduced before anesthesia. The cuff is inflated and anesthesia then may be induced in the usual manner. The cuff remains distended until the patient has fully reacted. Vomiting then occurs when the patient has regained all his protective reflexes. This is a new suggestion. Only time will tell how effective it is. It may be the answer to the problem of the patient with a full stomach.

Regurgitation and aspiration into the trachea may occur silently and unknown to the anesthetist. This has been demonstrated by studies in which dyes were placed in the stomach preoperatively and then identified in the gastric contents in the pharynx and the trachea. Berson and the writer working at the Charity Hospital in New Orleans introduced preoperatively into the stomach an insoluble dye, carmine red, which becomes soluble and red when made alkaline with ammonia. They noted that 15% of 1000 patients studied regurgitated the dye into the pharynx. In half of these, in other words 7%, the dye was identified in the trachea. The anesthetist was unaware of the regurgitation. The factors favoring regurgitation were as follows:

(1) *Difficult inductions.* The incidence was close to 25% in cases where the induction was labeled as stormy or difficult. Patients induced

with nitrous oxide ether sequence or ethylene ether sequence showed a higher incidence than those induced with cyclopropane or pentothal. Inductions were smoother with these latter agents. Obviously it requires considerable skill to induce a patient smoothly with nitrous oxide followed by ether.

(2) *The presence of the stomach tube.* The incidence was greater in patients who had Levine tubes in situ. This is probably explained by the fact that stomach tubes are usually employed in cases in which pyloric or intestinal obstruction is present and the possibility of dilated stomach and bowel is suspected. Besides the stomach tube does not always permit a snug fit of the mask on the face and induction may be difficult and stormy. It is advisable when a stomach tube is used during operation to open it entirely and aspirate the stomach contents with a syringe, prior to the induction of anesthesia. Furthermore the use of an intratracheal catheter with a cuff assures an even depth of anesthesia and a tight fit. If regurgitation occurs the cuff will protect against aspiration.

(3) *Intubated patients.* Intubated patients showed an incidence of regurgitation close to 25%. This was probably due to lightening of anesthesia after intubation. Also, patients who might possibly aspirate and had full stomachs were intubated more frequently.

(4) *Effect of position.* Patients who were in the head up position aspirated more frequently than those in the supine or head down position. In thyroidectomy, for example, all of which were intubated and which were performed in the head up feet down position, the incidence of regurgitation was 19%. All of these aspirated the dye into the trachea The number of patients in the head down position who aspirated was below the average. The supine head down position is preferred at all times when vomiting is anticipated. The incidence of regurgitation was above the average in the lateral prone and the prone position.

(5) *Influence of agents.* The incidence of regurgitation using pentothal and nitrous oxide contrary to our expectations was above the average of 15%. This was ascribed to the fact that pentothal is administered in fractions periodically when the patient lightens. The uneven maintenance of the desired level of anesthesia favors bucking and retching. The patient regurgitates when he lightens. The incidence was lowest when cyclopropane was used.

The amount of vomitus recovered in these studies was small, but, nonetheless, it was identifiable and indicated that an undesirable complication had occurred. In a later study of 117 patients in whom the value of the cuff versus the pack was assessed, it was noted that the cuff protected completely against aspiration. This study was carried out in the same manner with the same dye. No instance of aspiration into the trachea was noted when the cuff was used. On the other hand when the pack was used the incidence of regurgitation and aspiration was identical to that noted when the catheter was placed beneath the mask without a cuff or pack. In other words the pack affords no protection whatsoever in preventing aspiration of vomitus.

The statement has been made that fluid and vomitus cannot travel uphill. Obviously this statement is true but one must remember that vomitus can be sucked uphill. This is exactly what happens. The material is deposited in the pharynx in front of

the vocal cords and induces spasm. When the spasm is broken the patient takes a deeper breath than normal and the material is sucked upward into the bronchi by a powerful inspiration. Thus the head down position does not necessarily assure one that aspiration will not occur. When massive aspiration occurs the patient often develops profound shock, becomes comatose and dies within 24 hours. Lesser quantities of vomitus produce bronchiolitis. Lung abscess and pneumonitis are complications of aspiration. When small amounts are aspirated such as are seen in silent regurgitation, the body responds with slight degrees of atelectasis and a febrile reaction. Antibiotics were used routinely, in the postoperative period in the forementioned study. The incidence of respiratory complications due to the silent aspiration of small quantities of gastric contents was negligible.

Vomiting during the recovery period is often ascribed to anesthesia. However, many factors besides anesthesia are involved and anesthesia is only one of the many causative mechanisms. The effect of the drug on the medullary centers undoubtedly accounts for some of the vomiting. No doubt ether, cyclopropane and other anesthetics act this way in certain patients. There is little that the anesthetist can do to avoid this should a patient be susceptible to the drug. Fortunately not all patients possess this susceptibility. The use of anti-emetic drugs has been suggested preoperatively to obviate this but one does not know definitely whether they inhibit this type of vomiting. Some evidence exisits that the incidence of emesis is less in series in which patients were premedicated with anti-emetic drugs but the evidence is not conclusive. Anoxia during anesthesia is invariably followed by vomiting. The patient anesthetized with nitrous oxide who vomits and retches afterwards most likely has been subjected to anoxia. It is advisable at all times to fortify the gas with trichlorethylene or vinyl ether or use a basal narcosis of thiopental or premedication of morphine and atropine or scopolamine. Frequently pharyngeal and nasal airways, Levine tubes and secretions in the pharynx initiate vomiting by stimulating the pharyngeal reflex as the patient recovers and passes from stage III into stage II. One should remove airways as soon as possible after return of the pharyngeal reflex and aspirate the pharynx thoroughly to remove all mucous before the pharyngeal reflex returns. Often vomiting can be avoided by using this precaution. Once vomiting begins it seems to continue. Surgical manipulations, such as handling the intestines during exploration of the abdominal cavity and traction on the various mesenteries may be causative factors in postoperative nausea and vomiting. Peritoneal irritation which invariably occurs when the abdomen is opened and the peritoneum is incised may cause nausea and vomiting. Dehydration, acidosis, ketosis due to starvation and other factors attendant to surgery, such as electrolyte imbalance may be a causative factor. The medication used to control pain postoperatively particularly the narcotics, such as morphine and its allies may cause vomiting. Often, when patients who are under the influence of narcotics are moved, vomiting is initiated. The circulatory system is labile due to the influence of the drug and moments of hypotension develop which may produce cerebral anemia and vomiting. Thus, it behooves the anesthetist to determine what the cause of vomiting is before he attempts to treat this symptom.

The treatment of protracted vomiting in the postoperative period is symptomatic. A number of drugs are available which inhibit vomiting. The barbiturates, the anti-cholinergic drugs and the anti-histaminics are effective in many instances. For many years the combination of an anti-cholinergic drug, such as atropine or scopolamine together with a barbiturate, usually phenobarbital has formed the basis of sea-sick remedies. Frequently phenobarbital (1 gr.) intramuscularly is effective in sedating the vomiting center and curbing the vomiting. The incidence of vomiting in patients who have been premedicated with barbiturates or who have had thiobarbiturates during anesthesia appears to be less than those who have not had the drugs. The anti-histaminics such as dramamine or marazine may be used when phenobarbital is ineffective. It must be remembered that in certain patients these drugs cause sedation and hypnosis and overcome nausea and vomiting due to this effect. Instances of regurgitation while patients have been under the influence of anti-histaminic drugs have been reported. Thus they are not necessarily as effective as one might surmise. Chlorpromazine is one of the most serviceable of the anti-emetic drugs available at the moment. However, its use should be reserved for situations in which all other expedients have been tried and failed. The drug has undesirable side actions, the potential for causing severe and irreversible hypotension, the possibility of jaundice, skin sensitization and so on. To use chlorpromazine first is like using the heavy artillery to do a service that a rifle can do as effectively. In certain instances insertion of a stomach tube and lavage of the stomach with an alkaline solution, such as sodium bicarbonate, causes the gas to be expelled and removes ether tainted contents. In cases of dehydration distilled water and glucose intravenously frequently are effective and inhibit vomiting. Piridoxine has been suggested as an anti-emetic agent. The writer has been disappointed when it was used in cases of protracted postoperative vomiting.

Patients who are receiving narcotics develop postoperative vomiting when ambulated early. The centers responsible for compensatory circulatory adjustments when position is changed are depressed by narcotics. Presumably cerebral anemia develops. When the patient assumes the erect position a hypotension occurs. This vomiting is often ascribed to anesthesia. Vomiting which develops hours after the patient has recovered from anesthesia is due to some other cause and not anesthesia. The narcotic should be suspected if the patient is ambulatory and is receiving such medication.

The Geriatric Patient and Anesthesia

Richard H. Barrett, M.D., M. Sc. in Anes. *
Hanover, New Hampshire

This country can look with pride upon the achievements of American medicine, especially when we view the constant increase in the mean life span of our population.

Up until the past twenty-five or fifty years, many people spent most of their time looking for the fountain of youth.

At the time of the American Revolution the expectation of life at birth was 35.5 years; by 1900, it was 50 years; and in 1950 it had risen to 68.2 years.[1] It is even higher today.

Someone has said: "We have added years to life; now we must add life to years". In other words, we may not have found the fountain of youth, but the meaning of the term "old age" has certainly changed.

By definition, "Geriatrics" is that branch of medicine dealing with the diseases of the aged.

One of many things that have amazed me during the past ten or fifteen years is the number of cases of acute appendicitis which I have seen in patients over the age of sixty, or even seventy. Somewhere in my earlier training, I got the impression that appendicitis was a disease of young people.

When we consider geriatrics, are we concerned with the diseases—in the usual sense of the word—that older people may have, or, possibly, are we not comparing man with the automobile?

We have all seen automobiles that were manufactured fifty, or more, years ago which, today, run perfectly well. We have all seen men and women who now, at seventy, eighty, and yes, even ninety, years are in good health—and, I did not say "relatively good". They are in *actual* good health. We can postulate that the geriatric automobile may not have been used very much and if it were, it had good care. I wonder if the same can be said of men and women.

A few years ago I met a fellow, not as a patient, who made his home on Martha's Vineyard, off the coast of Massachusetts. His name was George Johnson, but everyone called him "Junior" or "Junie". He was 73 years old. And why did they call him Junior? You guessed it—in order to avoid mistaking him for his father. Junior had been married at least four times. All of his wives could have been listed in the "paraplenty" class. He started smoking at the age of 10 and drinking—whatever he could get his hands on—at 15, and, during his lifetime, he had not slighted either of these ventures. His occupation was "odd jobs" and some of them

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were mighty odd. The last time I saw "Junior" I would have said he was about middle age, whatever that is. To my knowledge, Junior has never been a patient.

Residents and interns sometimes start medical histories with this statement: "A white, married, *well-preserved* man of 45." Things like that can be irritating. What do they expect!

Not long ago we had a 75-year-old man in our hospital with intestinal obstruction. According to him, it was the first time he had ever been sick. I gave him a single dose, spinal anesthesia for this procedure, which proved to be a resection of about three feet of bowel. The operation required three hours to perform. When the dressing was being placed, I noted that the patient looked disturbed, so I asked him why he was scowling.

His reply was, "Damn it, young feller, you'd scowl, too, if you'd been layin' flat on your back as long as I have."

So I said, "Why don't you sit up?"

"Well, damn it, I didn't know I could," he replied.

"Well, damn it, you didn't ask me," I told him.

Whereupon he sat up, mumbling: "To think I lay there all that time and I could 'ave been sittin' ".

He looked a little uncomfortable with his feet stretched out in front of him on the operating table, so I asked him to try hanging his legs over the side.

This he did, appearing to be both pleased and disgusted with himself.

I asked if he wanted to walk back to his bed and he replied, "Been walkin' all my life. Can't see any reason to change now." He stepped to the floor. His knees buckled—as I expected they would; so, he accepted a ride in a wheel chair. Back at the ward, he refused to get into bed until he had, to use his words, "smoked a load" in his "T. D." I left him smoking his clay pipe.

Later I checked on him and the charge nurse told me that, after smoking his pipe, he got out of the wheel chair, walked about seventy-five yards to the bathroom, then came back to his bed and practically jumped into it, saying, "I guess I'll take a little nap." A geriatric case? Perhaps so. But, anyway, that *is* early ambulation.

From here on, I shall be telling you some of my impressions, some, or most of which, may be lacking in pure science, but I believe they have some practical value.

I shall cite few references simply because I wish to involve no one else.

Just where does the geriatric age begin? One writer in the field of anesthesiology says that we start deteriorating at the age of ten. Another writer, not in anesthesiology, but one who has written many bedtime stories would have me classed as being well "over the hump", for the past fifteen or twenty years. Still another author writes a medical article in which he says that the geriatric age begins at about fifty. Then, ten years later he writes another tome and—apparently having thought it over a little—states that old age starts at sixty, and I don't think he had been referring to life expectancy tables in the meantime.

Maybe we should be guided by the old adage that "one is as old or as young as one feels." A few trial starts are usually all that are necessary to prove this statement, one way or the other.

For the purpose of getting on with this discussion—and for satisfying

my own ego—I like to consider the geriatric class as being: all of those individuals who are older than I am and the adolescents are all younger—or, at least, they should be.

Anyone who is engaged in the administration of anesthesia is engaged in a dangerous profession. Every time you anesthetize a patient, the choice of life or death rests squarely in your hands and, it makes no difference whether you are a physician or a nurse. For this reason, it behooves all of us to know something about the people we are putting to sleep.

Let us consider that you are presented with a 75-year-old male who is wheezing and has an acute abdomen—possibly intestinal obstruction. What more would you like to know about this patient before giving him an anesthetic for that non-definitive surgical procedure know as "abdominal exploration"?

Yes . . . You would like to know his blood pressure. It is 180/90. You would probably like to have a chest x-ray, Possibly an electrocardiogram—a clinical graph which, as you may know, rules out nothing; a white blood count and a hemoglobin determination; an allergy history, especially if the chest x-ray and ECG are of no value to you. A little knowledge about his past surgical experiences may help. He has had none. You should also like to know if he has had ACTH or cortisone in the past two years. You get all of these things, and possibly more. Everything is within normal limits. Did you forget a blood volume determination, or have you looked in his mouth and seen that his tongue is abnormally dry and that his skin has lost its elasticity after being pinched? These are rough signs of dehydration.

If this man were 40 instead of 75, would it influence you in the choice of anesthetic method and drugs for the procedure at hand? I doubt if it would make much difference, in your choice of technique, but you would, or should, be influenced in the type and quantity of agents used.

In view of possible bronchial asthma you may elect to give "GOE"—nitrous oxide, oxygen, and ether sequence. Would you use pentothal or cyclopropane for induction? Possibly not, if you are versed in the pharmacological effect of these two drugs. Each is a parasympatheticomimetic drug potentially capable of producing laryngospasm, especially in an asthmatic patient. However, ask any anesthesiologist if he has ever given pentothal to a known asthmatic He has—probably to his chagrin—in some cases.

The whole point of all of this is that we are always dealing with an individual in the practice of medicine, and that includes the practice of anesthesiology, as well as surgery, internal medicine, or any other specialty or subspecialty. We rely upon findings that are applicable to a specific disease group—such as diabetes, arteriosclerosis, emphysema, cardiac insufficiency, but, in the final analysis,*it is always the individual* at hand.

Maybe you would prefer that our hypothetical case were to be given a spinal anesthetic. In choosing this type of anesthesia, or any other for that matter, are you apt to consider, first, why you should *not* use your method of choice rather than why you should?

Consider spinal anesthesia—it would give good muscle relaxation, reduce reflex stimulations, not necessarily involve respiration, provide a non-explosive field and several other advantages you could probably mention.

On the other hand—you have heard that spinal anesthesia is contradicted in intestinal obstruction. In theory, at least, the constriction of the bowel, created by the spinal anesthetic, could be sufficient to rupture it. I have never heard of such a thing happening. You may be dealing with a malignancy that had metastasized to the spine, or the route of injection of the spinal needle. The patient may be a diabetic or he may have a positive serology. If a physician gives an unknown (to the physician, at least) diabetic or luetic patient a spinal anesthetic and this patient starts walking with a steppage gait, a few months later, was the physician responsible for this added affliction? Probably not, but this fact might be difficult to prove to a lay jury. However, it is also well to remember that the well-being of a patient should not be jeopardized by failure to use an indicated anesthesia technique simply because of the possibility of litigation.

If spinal anesthesia were elected for the patient in question, would you prefer a single dose, or a continuous (or intermittent) dose technique? Would a vasoconstrictor drug be used intrathecally?

Let us assume that you have selected your anesthesia. Now, what about getting your patient ready for surgery? Remember, this patient may never have been in a hospital, but he has heard all kinds of stories about hospitals and anesthesia. When he was a "young fellow"—back twenty-five or thirty years ago—a hospital, at least to him, was a place where people went when they were about to die. However, hospitals, like everything else, have improved. But, he may not know this. Therefore—see your patient preoperatively — before he has had pre-anesthetic medication, preferably the day before surgery. If for no other reason than from the purely humanitarian standpoint, I urge that this visit be made by the anesthesia nurse as well as the anesthesiologist, especially if the latter is not yet available in your institution. Tell the patient what he can expect—both before and after anesthesia and surgery. Patients will frequently talk much more freely with you than they will with their surgeon. "Doctor so-and-so (the surgeon) seemed so busy, I didn't want to bother him" will be many a patient's excuse for not knowing more about what is in store for him. By some means, direct or otherwise, get over to the patient the fact—and that it should be—that he is not going to die under anesthesia.

Tell the patient what you are going to give him for anesthesia and approximately how you are going to give it. At least, tell him what the initial part of your procedure will be. If you have a post-anesthesia room or recovery room in your hospital, be sure to tell your patient that this is where he will be after surgery, so that when he awakens, he will not think he is in the wrong place.

Many older patients will have had one anesthetic, previously—such as that given for a tonsillectomy, in their youth. What an experience, and what a starting point in anticipation of another ordeal! Ask the patient about this previous anesthesia experience. He may tell you that he was strapped, sitting upright, in a chair, in a doctor's office. Then he was suffocated literally with ether on a mask placed over his face. He remembers having "passed-out", finally, and then came that awful "ether sickness" when, much to his surprise he was awake. Incidentally, it is well to remember that the so-

called "ether sickness" was due, probably, to hypoxia, not ether. Even so, ever since that experience, the odor of ether has always made him sick. You don't smell hypoxia; so, this sickness is blamed on ether. If Chanel No. 5 had been predominant during this early experience, that also might nauseate him today—maybe it does anyway. Association is the important factor.

Do not, for a minute, think that this patient is making up a good, or bad, story. He is absolutely right. Your responsibility is to make sure that, a year from now, he cannot tell a similar true story.

This type of patient is sometimes hard to convince. On occasion I have asked a patient how that 1910 car of his is running, and continued by reassuring him that, just as surely as he is not driving a 1910 automobile, we are not using 1910 anesthesia techniques.

Having convinced—or attempted to convince—this individual that he has a better chance of living during anesthesia and surgery, today, than he has while crossing the street, in front of the hospital, after his convalescence, you procede to order premedication, or, at least, check what others may have ordered for you. With the ever increasing popularity in the use of light anesthesia, for even the most major of surgical procedures, adequate premedication is more important than ever.

Do not be just a pair of hands. If someone else has ordered the premedication on the case you are going to do, be sure it is what you want for the patient you are going to anesthetize. You are a registered nurse, specially trained in anesthesia technology. You are about to embark on a life or death procedure, and it is expected that you will put to use all of the acumen that you have collected over the past several years of your life. If you do not agree with the premedication, or even the type of anesthesia, that has been ordered, by someone else, find out why it was ordered. It may be the best for the patient, but, be sure you know why. You are morally, if not legally, responsible for every patient you anesthetize. I am sure you understand what I am trying to tell you.

If you work with an anesthesiologist, your problems are reduced a hundredfold. If you do not work with an anesthesiologist, naturally, you will not emulate your surgeon or internist, but, you do have a right to know "whys and why-nots" of what you are trying to do.

Regardless of the type of anesthesia that is used, except for a very minor procedure, I like to have an 18-gauge needle in an easily accessible vein—nothing smaller than an 18. Frequently, I elect to have two 15-gauge needles well placed, governed by what I anticipate. This is based on the operation proposed, the condition of the patient, and, not least among these indications—the operator or surgeon concerned. It is usually preferable to place the needles in the forearm, thereby avoiding any contribution to possible phlebitis that might result if a lower extremity were used. The anticubital space should be avoided because of the necessary restriction in arm movement.

Always use procaine, or some similar local anesthetic, when introducing an 18-gauge needle—or any size, for that matter—unless the needle is passing through an already anesthetized area, as would be the case with a patient under general anesthesia. A slow drip infusion of 5% glucose in normal-saline will do the average patient no harm, and the needle is

readily available for supplementary medication or fluids, including blood. If you do not wish to give fluids, use a styletted needle, as would usually be the case if two 15-gauge needles were in place. Remember that the *sine quo non* of many a major surgical procedure has been an adequate venipuncture and, the best time to make that venipuncture is when you can find the veins.

Many a little "whiff of gas" or a "little shot of pentothal" has become a "weekend trip." As a matter of fact, I have often said that, in years past, we worried often about patients developing surgical shock on the operating table. Now we are apt to be more concerned about the possible ill effects of nostalgia.

In order to combat the dangers of this new entity (prolonged operating time) surgeons have taken to wrapping the legs of practically all patients, previous to surgery, in order to prevent venous phenomena. The presence, or absence, of these leg wrappings should be noted by you preoperatively. It is a part of the whole preoperative preparation.

With exactly what are we concerned in patients of the older age group? If they have painful arthritic conditions, we are careful in moving them and positioning them on the operating table. They may be very prone to develop pressure sores. They may be deaf. If you have not seen your patient before coming to surgery, you may never know this and, you may do him a great injustice and cause him much embarrassment.

What about premedication in older people? What result do we want from premedication? Regardless of the type of anesthesia to be used, we would like to have patients enjoy their trip to the operating room. Euphoria, under control, is a good thing at nearly any time, but it is especially valuable in the preoperative period. There is only one drug on which you can depend to produce euphoria consistently—that is morphine—but it is no respector of age.

Incidentally, if a patient should refuse to tell you her age you could give her a dose of morphine and predict it probably within about five years. Morphine affects a patient very closely according to his or her *chronological* age, not according to the age a patient may appear to be. That is, a cachectic 50-year-old who looks 80 will tolerate a given dose of morphine better than an 80-year-old who looks 50. The dosage used should be so regulated.

Scopolamine is an excellent drug and it can be given to patients even though they may be considerably older than I am. This is contrary to common belief. Also scopolamine can be used freely with patients of my age and younger—unless they happen to be chronic alcoholics, then I would recommend a little caution.

This brings us to another fine drug—a pharmacological depressant, pure and simple—but a social stimulant. It is my impression that alcohol is not used often enough, as a medication, in patients of my age and older—especially much older. Its action as a vasodilator makes it an excellent drug for producing relaxation in older people.

As you know, alcohol is dispensed in a 5% solution in 5% glucose and water as well as in several other combinations. In fact there is one combination on the market which contains alcohol, chlorides, other salts, sugar, vitamins, etc.—kind of an intravenous tossed salad!

The use of a liter of 5% alcohol preoperatively can do much to elevate

the morale of a depressed "tea-totaler" of any age, to say nothing of adding to his caloric intake. A liter of 5% alcohol is equivalent to 50 cc. of absolute alcohol. That is 50 cc. of 200-proof, or 100 cc. of 100-proof, or 150 cc. of the proof of most common brand whiskeys. Or to make it understandable to all of us—about a half a dozen Martini cocktails — very "dry".

When alcohol is used in the immediate postoperative period, it is possible to obtain good pain relief with this 5% solution, making it unnecessary to use any other analgesic, or to use analgesics only in minimal doses. Do not forget the vasodilating property of alcohol. It is one of the most valuable drugs we have for supportive treatment of the debilitated patient.

I have just mentioned one art of anesthesia. There is an art or science in anesthesiology which is losing ground rapidly. That is local, or block, anesthesia. There is hardly any surgical procedure that has not been, or cannot be, done under regional or block anesthesia in some form. By far the greatest amount of surgery is done under "local" or block anesthesia, in many other countries of the world. Far too often the contraindications to this type of anesthesia are that there are other cases pending or the possibility of a slight delay in starting surgery. But the most outstanding contraindications seem to be extra-mural in nature. Granted there must be someone available who knows how to do block anesthesia.

Many former patients, in the older age group, would be alive today had local, or block, anesthesia been used for that abdominal exploration, or that cholecystectomy, or that amputation, because adequately trained personnel administering potent anesthetic drugs were not available or not used. I shall not go into detail of the various anesthetic block procedures that can be used. Fortunately, several texts on this subject are now available. It was not always thus and, besides, most of you are not responsible for this type of anesthesia.

A few figures may be of interest:

Table I

MARY HITCHCOCK MEMORIAL HOSPITAL

Geriatric Surgical Cases

Age	1948	1953	1955
60-69	358	448	510
70-79	200	322	306
80-89	45	72	101
90-99	4	5	6

These geriatric patients, as a group, received spinal anesthesia and other forms of block and regional anesthesia, as well as various combinations of inhalation and intravenous anesthesia. Rectal anesthesia was not used in any of these cases. All of these patients had one or more surgical procedures.

The anesthetic technique, which, in our hands, for the past several years, has proved to be the safest for aged and debilitated patients is a combination of nitrous-oxide, oxygen and a muscle relaxant. This combination utilizes the well established analgesic properties of nitrous oxide. An adequate supply of oxygen administered by hand-assisted respiration prevents hypoxia. Any one of the curare-like drugs provides adequate relaxation of skeletal muscles. We have no real preference among the muscle-relaxant agents. We use succinylcholine, gallamine or d-tubocurarine.

Our technique is to start first an infusion of 5% glucose in quarter strength saline solution, in the adequately premedicated patient. We do

not hesitate to use scopolamine, even in the very aged.

Nitrous oxide and oxygen, in an 80-20 mixture, is administered for a few minutes by face mask. It is important to use non-rebreathing technique especially during the induction period, in order that bodily nitrogen will be replaced by nitrous oxide. Because nitrous oxide is a relatively mild analgesic, it is necessary to attain optimum concentration. The non-rebreathing technique also prevents build-up of carbon dioxide. After the patient is asleep, 20 to 40 milligrams of succinylcholine are given intravenously, through the intravenous tubing and the posterior pharynx and larynx are sprayed with a topical anesthetic solution. This spraying can be done before induction, but the comfort of the patient is not disturbed by delaying it until the patient is asleep. For that very short period while the muscle relaxant and the topical anesthetic are producing their optimum effects, administration of nitrous-oxide and oxygen is resumed by face mask.

A cuffed endotracheal tube is then inserted, under direct vision, and the patient is carried on hand-assisted respiration throughout most of the surgical procedure. This may be for one to eight hours or more. During the operation, muscle relaxant drugs are given as indicated. Occasionally small (5 to 10 mgm.) doses of meperidine (Demerol) may be given during the early stages of surgery; but, after that all maintenance is with nitrous oxide, oxygen and the muscle relaxant drugs.

Routine blood pressure, pulse, and often electrocardiographic tracings are followed. Intravenous fluids, including blood, are given as needed. On completion of surgery the patient is allowed to awaken gradually. If tracheobronchial toilet is necessary this is carried out while the patient is still relaxed, in order to avoid unnecessary coughing.

As the patient awakens he will usually open his eyes and respond to his spoken first name or nickname. The endotracheal tube is then removed. Straight oxygen is administered, for a couple of minutes, or longer, depending on circumstances. It is not uncommon, at this point, or during the short ride to the Post-anesthesia Room (Recovery Room), for the patient to ask for food or water. These cases rarely, if ever, vomit and, in many, nausea is looked upon as a postoperative complication.

All types of surgery can be and are done under this technique. Because of the safety of this method, many cases which, in years past, were declared inoperable, because of the age or physical condition or both, are now receiving the benefits of definitive surgery and being given a new lease on life. It is not uncommon to have patients on our surgical schedules who were declared inoperable 15 or 20 years ago—due to their age at that time, but especially to the absence of the muscle relaxant drugs. It must be remembered that the advent of muscle relaxant drugs has been a paramount factor in this technique. Decompensated cardiac patients, advanced asthmatics, markedly cachectic patients, and many others have received this type of anesthesia and many of them are still alive and well today. In our hands, there have been no cardiac arrests and no deaths using this technique.

One of my associates[1] has made carefully annotated observations—continuous electrocardiographic and electroencephalographic tracings as well as arterial oxygen and carbon

dioxide determinations—on over 150 poor risk cases between the ages of 60 and 95. The safety of the method has been well demonstrated. Patients are carried under very light anesthesia but in a relaxed and analgesic state. In this condition, the natural homeostatic defense mechanisms of the body may function freely because they are not hampered by the depressant action of deep anesthesia. Untoward reflex phenomena from abdominal and pelvic viscera have been much less common under this light form of anesthesia than under deeper stages produced by other more potent agents.

Thiopental (Pentothal Sodium)® in small doses, is used occasionally with this technique, as an induction agent, in even markedly debilitated patients. However, experience has shown, in these cases, that even small doses of this drug may cause potentially harmful lowering of blood pressure.

Ethyl ether, in relatively low concentrations, may produce equally as harmful depression of blood pressure—even to the shock level. However, it has been demonstrated elsewhere[2] that, in certain types of cases, very light planes of ether anesthesia are innocuous.

The technique of light anesthesia, produced by nitrous oxide and muscle relaxant drugs, provides the very definite advantage of a nearly wide-awake patient at the completion of surgery. Active hyperventilation and coughing, when indicated, can be instituted immediately.

There is no residual respiratory depression. Postoperative analgesics can be given as actually needed, rather than as estimated. Postoperative care is greatly facilitated.

I would like to say just a little more about analgesia, especially as applied to older people. There is one analgesic drug that, in many areas, is being used less and less. I refer to morphine. We should remember that the one natural antidote to morphine is pain. Therefore if a patient is in pain morphine can be administered safely to the point of obtunding pain without causing any respiratory depression. Trial doses may be given intravenously in dilute solution, by a physician. It will be found that many older patients will have complete relief from pain after the administration of only three or four milligrams of this narcotic. The test dose will give a fairly accurate idea of the amount to be ordered for future administration by a nurse.

I have seen too many patients suffering needlessly, simply because a routine system of ordering meperidine (Demerol) or some other morphine substitute happens to be the order of the day. Economically it is unsound to use the so-called morphine substitutes. Pharmacologically it often borders on sheer cruelty, due to the inadequacy of dosage prescribed. In the use of morphine, it should be remembered that its optimum effect is realized in twenty minutes or less, when it is given intravenously, and in about one hour and a half when it is administered by the subcutaneous route. If you are dealing with a patient who is cold from exposure or from traumatic shock, or one who has generalized peripheral vasoconstriction, from any other cause, practically any medication given subcutaneously may have no effect for several hours and then, only when the vasoconstriction is released. Morphine like water can be very useful. A little can be of great value, but no one benefits by a flood.

SUMMARY

There are many other things that we could talk about in relation to geriatric anesthesia and pain relief but we must stop somewhere.

In closing, may I leave you with these thoughts—the practice of geriatric anesthesia today in any general surgical hospital is the practice of clinical anesthesia per sé; and the practice of anesthesia itself, as a specialty, is, and always has been not the specialized knowledge of what to do now, but rather, the acumen gained by study and experience which qualifies one to know *what to do next.*

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The American Association of Nurse Anesthetists and Hospitals

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This is the 25th anniversary of your Association. You are to be complimented on an achievement of considerable magnitude. The importance of your Association and of its task is so great that to use flowery language in discussing it would be undesirable and serve no useful purpose. Rather, the presentation of important facts and trends in simple, concrete language expressed in a manner that can be understood is essential. We hope in a measure to be able to do just that.

The history of the organization of your Association is unique. The uniqueness lies in the fact that your organization was developed because of hospitals and not separately, as was the League for Nursing and the American Nurses' Association. This is fortunate. The destiny of the nurse anesthetist and hospitals are closely associated. For centuries hospitals provided most services except diagnosis and therapy—in other words, a sanctuary, food, shelter, nursing care, hot baths, music and sympathetic care—everything but a cure. Now, advances in scientific medicine have provided hospitals with newer and better opportunities for service to ailing humanity. It is significant that these advances are the ability to diagnose and cure many diseases and conditions which, through the centuries, were not diagnosable or curable. One of the most important forms of therapy is modern surgery, and it goes without saying that modern surgery cannot be performed without anesthesia. We go further and say that modern surgery cannot advance without a parallel advance in anesthesia. We have developed a surgical approach to most parts of the human body, but to illustrate our point, cardiac, thoracic, artery, and brain surgery cannot advance without a simultaneous development in anesthesia. One by one we are discovering and perfecting new anesthetic agents and technics for these surgical procedures, but there are still serious gaps in our knowledge, so that it is most important that the science and art of surgery work closely with the science and art of anesthesia. Otherwise, advances in surgery will be retarded.

The motivation behind the organization of nurse anesthetists, as far as the record and our experience can determine, came from two sources. One, the persistent attempt by pressures from physician anesthetists to legislate the nurse anesthetists out of existence; and two, the action of the American Nurses' Association in sponsoring a movement to consolidate office nursing and nurse anesthesia under the nursing department and not as a hospital service under the

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guidance of the surgeon. In 1930 the nursing leaders must have thought nurse anesthetists were unimportant numerically and functionally. To group nurse anesthetists with office nurses overlooked the hospital entirely. In other words, the American Nurses' Association and the League for Nursing lacked vision in not seeing the possibility of a nursing specialty in anesthesia as a vital function essential to the growth of scientific surgery and the modern general hospital.

The American Association of Nurse Anesthetists was organized on June 17, 1931 in the classroom of the Department of Anesthesiology at Lakeside Hospital, Cleveland, Ohio, between the hours of 2:30 and 5:30 p.m. Your organization, at that time, was called the International Association of Nurse Anesthetists, but before the Articles of Incorporation had gone through, the name was changed to the National Association of Nurse Anesthetists rather than International. On March 12, 1932, the National Association of Nurse Anesthetists was incorporated in the state of Ohio.

The new organization now sought affiliation with the American Nurses' Association when Agatha Hodgins applied by letter on November 16, 1931. On April 9, 1932, the American Nurses' Association voted that the National Association of Nurse Anesthetists could not be accepted for affiliation. This left no alternative but for your Association to be a distinct entity. It also closed the door to the possibility of meeting concurrently with the American Nurses' Association. It was only natural that the first meeting of the American Association of Nurse Anesthetists was held concurrently with the American Hospital Association which met at Milwaukee in September, 1933. The American Hospital Association had the vision and good sense to welcome the nurse anesthetists in the belief that meeting concurrently would have many mutual benefits which both Associations would enjoy. Experience has proven the mutual pleasure and benefit, for the arrangement is still in effect and we trust that it will continue far into the future.

We have said that the League for Nursing developed separately from hospitals. This statement is not intended to be critical of the League but to explain the lack of interest on the part of nursing organizations in hospitals, hospital nursing and nurse anesthesia. It is a historical fact that the League was organized in 1893 before the modern hospital was more than a dream. The Johns Hopkins Hospital, one of the first modern hospitals, opened in 1898, five years after the League was organized. The modern hospital was not built in large numbers until the 1920's and was the natural result of the advance in scientific medicine. For the first time medical schools had something to teach and a place in which to teach the art and science of medicine. The internship and residency in medicine was made possible.

We believe the fact that the League for Nursing was organized apart from medicine and hospitals has made it difficult for nursing leaders to have an interest in hospitals and therapeutic medicine. Lack of interest meant lack of knowledge and nursing education has not followed the pattern of medical education in that it uses the hospital as a college for two years of undergraduate medical education. the clinical clerkship. The hospital is the college for all of graduate medical education, the internship and residency. Nursing education has not

yet developed the internship, and does not share the view of the American Medical Association and American Hospital Association that the hospital is the proper college for graduate medical and nursing education.

This lack of interest in the hospital has led to a positive stand by many nursing leaders that schools of nursing should be separated from the hospitals and placed in universities and colleges. It is difficult to understand why in suggesting a collegiate program for nursing education that there was no suggestion of offering a B.S. degree for training in nurse anesthesia. So far, to our knowledge no school of nursing, diploma or collegiate, is teaching the nurse anesthetist. All schools for nurse anesthetists are hospital schools.

The advances in surgery and anesthesia have necessitated a change in the education and training of the nurse anesthetist. The training period has been lengthened, and physician anesthetists who are developing new technics and concept of patient physiology not only in cardiac, thoracic, general, and neurosurgery but especially in pediatric and geriatric anesthesia, are needed to teach the nurse anesthetist. Formerly, with the lack of interest of the medical anesthetist, much of the teaching of anesthesia to the nurse anesthetist was done by leading surgeons, particularly those who were pioneering in the new fields of surgery. But the advances of scientific anesthesia are so varied and technical that the surgeon no longer has either the time or the ability in many instances to teach the nurse anesthetist. We are, then, confronted with the necessity of getting the cooperation and understanding of the medical anesthetist in engaging in the education of the nurse anesthetist. This has been a slow and difficult task, but the earnest efforts of many surgeons and hospital administrators have been successful in obtaining a significant number of medical anesthetists to perform this important task. Furthermore, your efforts as an Association and as a profession in illiciting the interest and cooperation of the medical anesthetists has also been successful. This fact is proven with each issue of your journal, because you will see many contributions by medical anesthetists. There is an increasing number of hospitals which have a dual program of the training of medical residents in anesthesia on one hand and nurse anesthetists on the other. This is a forward looking situation and must continue if the surgery performed in our modern hospitals is to be in the volume which the community needs require. In our opinion, we feel that a chief or administrative nurse anesthetist should be delegated the responsibility by the medical anesthetist for the routine training of the student nurse anesthetist and for routine administration of anesthesia in the hospital. This leaves the medical anesthetist more time for teaching, research, and development of new technics. The medical anesthetist should be the liaison between physiology, pharmacology, and applied anesthesia. This is essential for research, for testing new agents, and for developing new technics particularly for the newer surgery. He can give the student nurse anesthetist better theoretical training. In addition to training residents in anesthesia, he is in position to train interns and residents from other services who rotate for a period of time through anesthesia. We consider this a very important function of the medical anesthetist for the reason that physicians, particularly surgeons, should have a basic understanding of the

fundamentals of anesthesia. If the hospital is affiliated with a medical school, medical students are taught.

Your Association is to be highly commended for having taken a courageous step and set up an accreditation program for nurse anesthetists. This has been of great significance, has raised the standard of training, and has given your organization stature and authority. Anesthesia is a specialty that requires long training. The science of anesthesia to a considerable extent can be taught, but the art of anesthesia must be practiced until the anesthetist has the know-how, the skill, the feel, and the equanimity that can come only with experience, care, and skill of the anesthetist.

It behooves us to have a sufficient number of good schools turning out nurse anesthetists in adequate number. While this may be a competitive action on the part of the nurse anesthetists to combat competitive action of medical anesthetists, competition is thoroughly understandable and as far as the public is concerned and in our free enterprise system, is desirable. You as an Association need not be too alarmed over these challenges if you and your members will abide by the general principles we have laid down. The trend of medical practice, the advance of medical science including anesthesia, the trend of public reaction towards medical care indicate definitely that the hospital is the center of medical care, and that care cannot be given without sufficient anesthetists. If you continue to give good service of high quality at reasonable cost to patients, if your schools of anesthesia maintain standards and number of students, you will have gone a long way in meeting the challenge.

There must be in the modern hospital the greatest collaboration on the part of the nurse anesthetists with the medical anesthetists and the operating surgeon. She must have the ability to work with the intern and resident staff, and this is important, to cooperate with and attain the cooperation of the nursing service of the hospital. The nurse anesthetist needs the good will and assistance of all these groups, and considerable effort should be made by you as individuals and by your organization to gain this good will and understanding of other groups in order to help you meet the challenge of today. You already have the understanding and cooperation of the hospital administration, and we must work with you in every way possible.

Your Association is fortunate in having Florence McQuillen as your Executive Director. Your officers and trustees indicate that they understand the problems and are willing and capable of solving them. Your Association is cognizant of the challenge to the nurse anesthetist, which is the overall challenge in a normal life history of a profession and its organization — birth, growth, production, reproduction, maturity, and death. The challenge of death highlights one of the very useful functions of an organization. Organizations with their large numbers of individuals of active age groups provide a continuity which bridges over the death of individual persons and technics.

With our needs and destiny so interwoven, hospitals and their surgical staff have shown great interest in nurse anesthesia. There is no other way to provide anesthesia in volume because there are not enough medical anesthetists available. There is still a shortage of medical anesthetists, and that condition will continue in

the foreseeable future. This makes the place of the nurse anesthetist very secure.

We cannot close this discussion without mentioning the names of some of the founders and leaders of your organization. We need to cherish the memory of those individuals who preceded us and laid on broad lines the foundation upon which the organization is built. Year by year the memory of these individuals fades and the shadow of oblivion falls deeper until a name alone serves a link of continuity. Let us recall only a few of them . . . *Agatha Hodgins**, *Gertrude Fife, Helen Lamb, Verna Rice, Aida Aldwein, Sister Alexandrine**, *Miriam G. Shupp, Dr. George Crile**, *Dr. Evarts Graham, Dr. Robert Bishop, Jr.**, *John Mannix, Dr. Burt Caldwell**, then secretary of the American Hospital Association, *Dr. Malcolm T. MacEachern**, *the Mayo brothers**. There are many other famous names on the roles of this organization that time does not permit us to recall. In these hectic days it is difficult to maintain the remembrance of individuals who made possible the present with its prospects and the future with its possibilities. Therefore, we mention some of them to gain inspiration from their actions and to maintain a sense of historical continuity. Historical continuity serves to preserve basic principles, and a review of the historical past of this great Association adds new interest and information to a basic fact—that the nurse anesthetist is essential to modern surgery and the modern hospital.

*deased.

The Prevention, Recognition and Treatment of Postoperative Atelectasis

Paul A. Clayton, M.D.* Salt Lake City, Utah

Pulmonary complications are common following major surgery, varying in incidence from two to six per cent. Among the factors involved in producing these complications are: 1. Reduction of the Volume of Respiration.--This may be as much as fifty per cent and may be due to poor anesthetic management, pain, too tight dressings, and too deep anesthesia or over-sedation; 2. Type of Patient.--Complications are more frequent in diabetics, decompensated cardiacs, and obese patients; 3. Duration of Anesthesia.--Complications are more frequent in prolonged procedures. 4. Type of Operation.--Chest and upper abdominal procedures are more prone to produce pulmonary complications.

The most common of all postoperative pulmonary complications is atelectasis. Let us briefly consider the pathogenesis of atelectasis. Two common theories are used to explain its development. In the first it is felt that a mucous plug of thick secretions forms in a bronchiole shutting off the alveoli distal to the plug. The air in the plugged off section is gradually absorbed causing a collapse of the area involved. In the second theory a membrane is formed across the lumen of a bronchiole. This membrane is slowly moved forward by ciliary action causing a suction like action on the alveoli distal to the membrane causing their collapse. In either case mucous secretions are involved. Foreign bodies of any type could produce the same plugging effect.

Prevention of the disease is always better than treatment if prevention is possible. In prevention of atelectasis, recognition of the probable cause is the first step. Let us make a more thorough break down of some of the before-mentioned factors involved in the recognition of the probable case. First, is the type of operation involved? Chest surgery is notorious because of the very nature of the work. Holding part of a lung in collapse tends to promote atelectasis. The longer the lung is collapsed at any one period, the more difficult it is to maintain the lung in its normal inflated state after it is reinflated. That is the reason it is common procedure to periodically reinflate the collapsed portion of a lung during a chest operation. Upper abdominal surgery such as stomach and gallbladder operations is more prone to produce atelectasis than is lower abdominal surgery. Lower abdominal surgery especially with prolonged Trendelenberg position, produces

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more atelectasis than lower extremity surgery. Repair of large ventral hernias must always be considered as a possible producer of atelectasis. Falconer[1] reported a very high percentage in a series of kidney cases done under spinal anesthesia.

Patients in whom atelectasis is apt to occur may also be classified as to body build. The obese patient, the patient with the short thick body, and the barrel chested patient are more apt to be involved than the tall thin patient.

Pathological conditions predisposing to atelectasis can be listed, such as bronchiectasis, chronic bronchitis, respiratory obstruction from any cause, and any condition promoting excessive secretions, such as chronic sinusitis, poor oral hygiene, and recent or acute upper respiratory infections.

The importance of excessive mucous or secretions cannot be overemphasized. The preoperative visit or check before giving an anesthetic can be of extreme importance in this respect. Ask the patient to cough and listen carefully for the "wet cough". If it is present, a few questions concerning excessive smoking, postnasal drip, a recent cold, or large quantities of sputum raised may immediately put you on guard as to how the patient should be handled. If a patient with a "wet cough" is to have one of the before mentioned operations, you must use all your knowledge and skill to prevent the almost certain postoperative chest complication.

The skill of the anesthetist is of primary importance as compared to the type of agent used. We still hear from our patients about "ether pneumonia" and how they nearly died. The ether had little to do with it and a much better name would have been postoperative atelectasis. The choice of drug or agent is not of major importance but the skill with which it is used is. Atelectasis may be far more frequent on some services under spinal anesthesia where nothing but air or oxygen is put into the lungs. Paralyzing the intercostal muscles, whether it be with spinal, deep ether or cyclopropane, or curare, has the same effect. It decreases ventilation and if allowed to persist promotes incomplete distention, which is Collins'[2] definition of atelectasis. So, let us not blame the agent we are sometimes forced to use for the difficulties arising postoperatively.

Let us talk a little about the actual management of the anesthetic from the standpoint of preventing atelectasis. Most of you are undoubtedly in institutions where the surgeon dictates the choice of the anesthetic to be used, and you are placed in the position of keeping him happy in his choice. If you are in an "ether" institution because of a ruling made due to some unfortunate cyclopropane explosion, just remember that the number of postoperative chest complications will vary directly with the amount of third to fourth plane anesthesia used. Learn to know your surgeons and the points during an operation when he will demand maximum relaxation. Learn to carry the patient in a lower first plane or upper second for the greater part of the time. Anticipate ahead of time when you have to deepen the anesthetic, and he won't criticize you for carrying the patient too light the balance of the time. Learn to support and deepen and smooth out respirations in the third plane without deepening the anesthetic still further, and in this way help to maintain adequate lung expansion during those periods when the surgeons say, "Keep

him down".

If you have learned to use curare with ether, supported respirations are a must if you would avoid the inadequate lung expansion and the carbon dioxide build-up that occurs with it. With the use of cyclopropane and curare, pentothal and curare, or any such combinations, controlled or assisted respirations are a must if you would avoid the same trap of inadequate lung expansion It is a frightening experience to me to walk into an operating room and see a mere flutter of the breathing bag with the anesthetist very intent on checking the blood pressure or pulse. All I can say is keep those lungs well ventilated. It is much easier to keep them well expanded during surgery than to try and re-expand them when the operation is over.

Although you must go along with your surgeon in the use of accepted operating positions, don't be overzealous yourself in the extreme use of the kidney and gallbladder rests, sand bags to distort the normal position of organs, etc., especially in the normal or easy case. These may be necessary crutches at times but as far as chest ventilation is concerned they are instruments of torture.

Learn to be skillful enough with whatever agent you use, to have the patient awake at the end of the procedure, so that if you ask him to take a deep breath he will do so, and if you ask him to cough, he at least makes the attempt. If you can do that, the surgeon will not only think you are wonderful, but the floor nurses will count you as their best friend.

Oxygen Determination

There is a common practice in finishing an anesthetic that I have always been opposed to. That is the practice of waking a patient up on one hundred per cent oxygen. Oxygen is not only a physiological gas but it is absorbed from the lungs in a manner unlike any of the other gases. All other gases both on the gas machine and in the air obey the gas laws. That is, they are absorbed or diffuse across the alveolar membrane according to the laws of partial pressures and come into equilibrium on both sides of the membrane. This is not true of oxygen in the same sense due to the tremendous power of the hemoglobin to combine with oxygen. As a result of this power, oxygen is transported across the membrane in much larger amounts and in much less time than the other gases. In simple English, this means that if you fill the lungs with pure oxygen unmixed with some other inert gas such as nitrogen, which forms almost eighty per cent of the air we breathe, or helium which is to be found on most gas machines, or nitrous oxide which is on every gas machine, this oxygen undiluted in the alveoli will be quickly absorbed and when you take the mask off, if the patient is not breathing deeply, there is no rush of inert gas such as nitrogen from the air, to all parts of the lungs to keep the alveoli distended. Those remote parts of the lungs not reached by the first air inhaled may have all the oxygen absorbed from them and become collapsed because there is no inert gas to keep them distended. It is far better, in my opinion, to use a fifty-fifty mixture of nitrous oxide and oxygen, if helium is not available, to terminate an anesthetic than to use one hundred per cent oxygen.

Tracheobroncheal Toilet

We are now at the end of the operation and here one of the most important steps in prevention occurs. If the intratracheal tube has been

used, the job is easy. I am referring of course, to the tracheobronchial toilet. The agents we have used, the airways, the intratracheal tube, possibly a Levine tube, are all foreign bodies and as such produce some reaction. The most common reaction is that of mucous production. The nasal passages, the mouth and the oropharynx should be carefully suctioned. This removes dangeorous secretions that might later gravitate downward into the lungs to plug a bronchus. If an intratracheal tube is in place, it is an easy matter to pass a catheter through the tube and suction the trachea and by manipulating the tube, the right and left main bronchi. This has the added advantage of stimulating a vigorous cough so that mucous far down in the bronchial tree, beyond the reach of the catheter, will be coughed up to a point where the catheter can pick it up. If no tube is in place, by careful manipulation of an airway, with slight flexion of the head, the catheter can often be made to go between the cords into the trachea and a tracheal toilet can be accomplished. Removal of accumulated secretions at this time when the patient is not yet able to get rid of them himself is one of the most important steps in prevention of chest complications. Nothing else can take its place.

Postoperative Treatment

Although you may have nothing to do with the postoperative orders, you must realize that the first postoperative hypo may have a lot to do with whether your patient is going to have trouble or not. The patient who is allowed to completely regain consciousness and roll from side to side in bed, is far better off than the one who is given a hypo as soon as he is back in bed so that he won't get too restless and cause the floor nurse a lot of trouble. You can just about depend on it that the patient who lies like a log for several hours will have to be worked on to keep his lungs clear.

Postoperative carbon dioxide-oxygen inhalations, frequent turnings, chest pounding, and encouragement to cough, are all very important to the prevention scheme. But the way you handle the patient during the anesthetic and immediately on its termination are far more important. On our own service, we have cut the incidence of atelectasis way down by careful attention to the details I have mentioned. The old adage is still true, an ounce of prevention is worth a pound of cure.

Conclusion

We have discussed prevention of atelectasis, but in spite of our efforts it is still a complication we must occasionally contend with. What can you as nurse anesthetists do to help? I assume you are all in the habit of checking your patients on postoperative visits. If atelectasis is not recognized as an immediate complication of the surgery and anesthetic, it characteristically appears in about forty-eight hours. Pain is not common but a desire to cough is. Evidence of oxygen want is usually present such as some degree of cyanosis, shortness of breath, labored respirations, and an increase in pulse. Any patient with a temperature of 101 or above, must be considered as a suspect. The most important sign is asymetric chest movement. Usually with such a patient, if treated immediately, there is not too much trouble and results are good. With the patient turned with the affected side up, a little pounding on the chest over the involved area may be enough to dislodge the mucous, cause it to move,

and thus stimulate a coughing spell. Deep breathing with a good cough may do the trick. If the patient is uncooperative and refuses to cough due to pain, supporting the incision with one hand and the other placed behind the back makes it easier. Sometimes a visit just after a hypo will find the patient feeling more comfortable and he will be more in the mood to cooperate. If these efforts are not sufficient, carbon dioxide-oxygen inhalations may stimulate breathing sufficiently to move the mucous slightly and thus set up a coughing spell. It may be necessary to resort to all these maneuvers.

If the patient is semi-comatose and unable to cooperate, it may be necessary to pass a suction catheter into the trachea, which always sets up coughing and at the same time the secretions can be sucked out. We have found that in such patients we have more success in getting the catheter into the trachea, if we use a large Guedel airway in the mouth and pass the catheter through this. By moving the airway gently and turning it slightly until the right angle is found we have had close to one hundred per cent success in passing the catheter between the cords into the trachea. After the catheter is in the trachea, turning the head slightly to the left will usually cause it to pass easily into the right main bronchus. This is the easiest side to get into and most cases of atelectasis occur on the right. By turning the head to the right, it is possible with some experience to get into the left main bronchus. This procedure can often be life saving, especially if you do not have a good bronchoscopist available.

I realize that such procedures rarely fall to your hands but some of you may be working where the services of a specialist are not available, and your knowledge of airways and their maintenance is greater than any one elses due to the fact that you are working with them every day. To you especially some of these techniques may prove of great value and a life saving procedure.

Cortisone and Anesthesia

Sherwood W. Gorens, M.D.*
Wood, Wisconsin

The subject of my talk is the Surgical and Anesthetic problems arising from the clinical use of cortisone.

Before I delve into a consideration of the problems created I thought it would be well to consider briefly: (1) The theoretical and fundamental concepts of the pituitary adrenal relationship normally and with stress; (2) How the clinical use of cortisone affects this relationship, and (3) The major Surgical and Anesthetic problems arising from this altered relationship induced by the clinical use of cortisone and the management of these problems.

Certain terms will come up frequently during the course of this talk and I would like to pause for a definition of these terms.

Selye has introduced the terms *stress* and alarm *reaction*. A stress or stress agent is any physical, chemical, bacterial, environmental or psychic change or agent which produces a reaction of adjustment. A few examples of stress agents are (1) Cold, (2) Infection, (3) Anoxia, (4) Anesthesia, (5) Surgery. The response to stress is mediated thru a pituitary-adrenal interrelationship. Selye has applied the term "alarm reaction" to the sum of all the physiological and biological responses and phenomena elicited by the exposure to stress agents or stimuli.

What are steroid hormones? They are a group of compounds that resemble cholesterol, having the same ring structure—a cyclopentanophenanthrene nucleus. All cortical hormones are chemically very closely related to cholesterol and hence are sterol like or steroid hormones.

In all probability the theories I am going to present will be modified in the future as newer experimental data are accumulated. This report will deal with the present concepts.

First, let us consider the normal pituitary adrenal relationship (Figure 1) There is a humoral substance from the hypothalamus that acts as a trophic agent to the pituitary and enables it to maintain its normal production of adrenocorticotrophic hormone (ACTH). The anterior pituitary secretes ACTH which stimulates the adrenal cortex to elaborate all adrenal cortical steroid hormones. The cortical hormones fall into four main functional groups. (Figure 2) It should be noted that these steroids have effects in more than one of these four categories. They have pronounced effects in one of the major categories and minor effects in others.

Now after the stimulation of the adrenal cortex by ACTH the blood level of cortisone and cortisone-like

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substances called corticoids increases and further production of ACTH is inhibited correspondingly if the blood level of corticoids decreases there is increased production of ACTH.

So much for the normal relationship. Let us next examine the emergency mechanism set into operation with stress—say the stress of anesthesia and surgery. (Figure 3) With the stress there is a release of epinephrine from the adrenal medulla and there are afferent nerve impulses both of which reach the hypothalamus. These stimulate elaboration of a humoral substance which causes the pituitary to release greater amounts of ACTH. The speed of ACTH release after stress is very rapid. It has been shown to be less than *10 seconds*. Within seconds then—*if the adrenals are normal* you get a tremendous rise in the output of adrenal hormones and corticoid steroids and a rapid adjustment is made to the stress situation. If the individual is to make an effective adjustment to stress situations, he must have an intact, normally functioning and responding adrenal gland to perform emergency adjustments. Now if there is adrenal insufficiency or adrenal

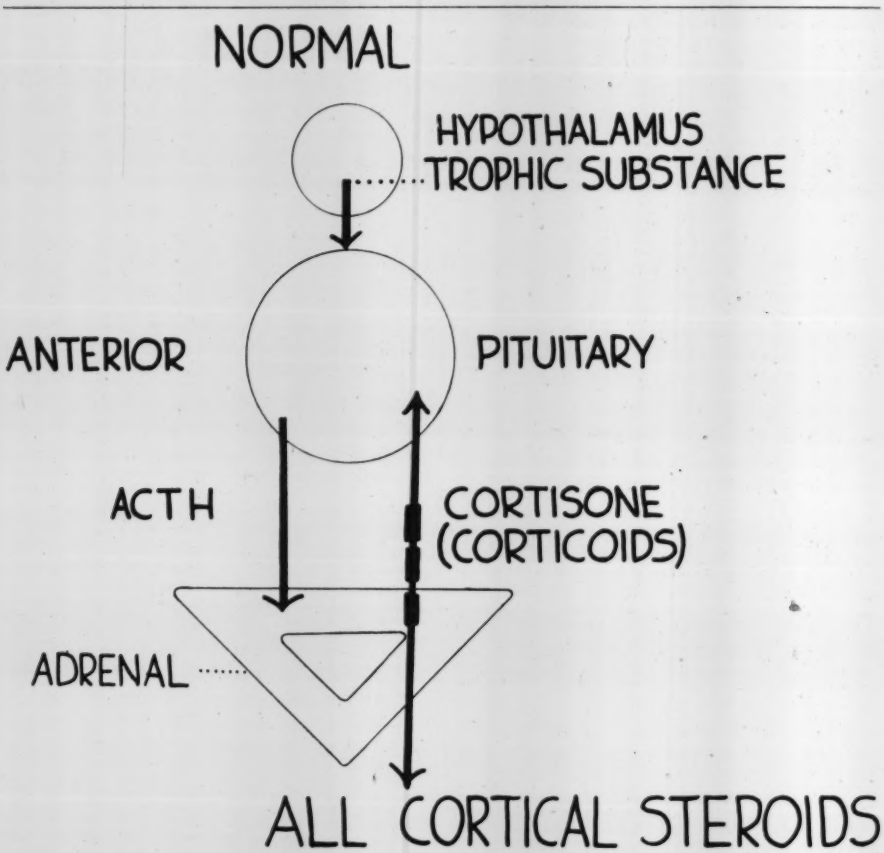

Figure 1

atrophy so that with stress there is little or *no* adrenal response the patient will go into shock with all its manifold manifestations — tachycardia, fall in blood pressure, decreased body temperature, pallor, decreased muscle tone, hemoconcentration, anuria, etc.

Now I come to the second part of my talk.—How the clinical use of cortisone affects the pituitary adrenal relationship.

It is a fundamental law of endocrinology that when a potent hormone is administered in doses that equal or exceed the physiologic requirement, the function of the gland in which the hormone normally originates is suppressed and there is varying degrees of cytologic change and atrophy. It has long been known that following administration of dessicated thyroid orally, thyroid function is depressed and normal function may not return for 1 to 2 months, that 3 to 6 months may be required for the testes to recover after the prolonged use of testosterone. The adrenal gland is no exception to this fundamental law. If exogenous cortisone is administered, there is suppression of activity of the adrenal cortex and you get adrenal insufficiency and atrophy. Adrenal atrophy can occur with as little as 5 days of cortisone and with as small a dose as 20 mg/day. When cortisone is given for more than 5 days the reduction in adrenal weight is constant.

Evidence exists to indicate that with more prolonged administration of cortisone, suppression of adrenal cortical function may persist for as long as 3 to 6 months after the use of the hormone is discontinued. However, in most cases it is believed that adrenal function is back to normal 6 weeks after cortisone is discontinued.

FOUR FUNCTIONAL GROUPS OF HORMONES

1. ELECTROLYTE METABOLISM; Na, K, Cl, AND H_2O.
   (DESOXYCORTICOSTERONE)
2. "S" OR CBHY. & PROTEIN REGULATING STEROIDS
   (CORTISONE AND OTHER CORTICOIDS)
3. ANDROGENS, ESTROGENS AND PROGESTERONES
   (a) SECONDARY SEX CHARACTERISTICS
   (b) NITROGEN RETAINING
4. PIGMENT STEROIDS

Figure 2

It is inescapable that exogenous cortisone can produce adrenal insufficiency and atrophy. This is of tremendous significance since we know there must be an intact adrenal in meeting the physiologic demands of stress situations such as anesthesia and surgery. It is of great import because of the number of patients who have already received and who will receive cortisone and who may have potential adrenal insufficiency.

There are several million arthritics alone who have or will receive cortisone. In addition, cortisone has been

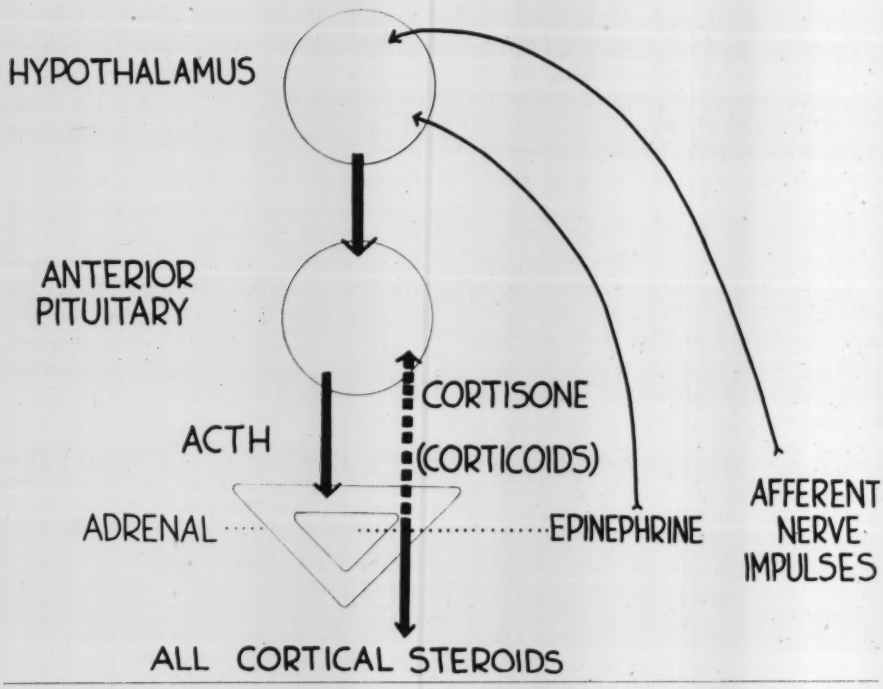

Figure 3

recommended and used in acute rheumatic fever, lupus erythematosis, ulcerative colitis, hemolytic anemias, uveitis, iritis, psoriasis, dermatitis, urticaria and so on.

It is apparent that many children and adults have or will have received cortisone and may have potential adrenal insufficiency. The patients will get along well under normal situations but if any of these patients are subjected to the stress of anesthsia and surgery the adrenal is unable to respond to the emergency situation by release of steroid hormones and the patient may go into a state of shock and may die. Three deaths following anesthesia and surgery in patients with cortisone induced adrenal insufficiency have already been reported in the literature. Let me present one near catastrophe that occurred on our service at Wood.

Case History (Figure 4)
27 year old, colored male, with ulcerative colitis diagnosed 2/23/-53 and treated with cortisone the last time for one week three weeks prior to surgery.

Now recall that a patient may develop adrenal insufficiency for stress situations with as little as 5 days of cortisone and that with more prolonged administration the likelihood is very much greater and that this suppression may last for 3 to 6 months. It would be expected then that this patient would have adrenal insufficiency with the stress of anesthesia and/or surgery. Figure 4 shows what happened.

The large number of conditions for which cortisone is employed and the large number of patients to whom it is given represent an important

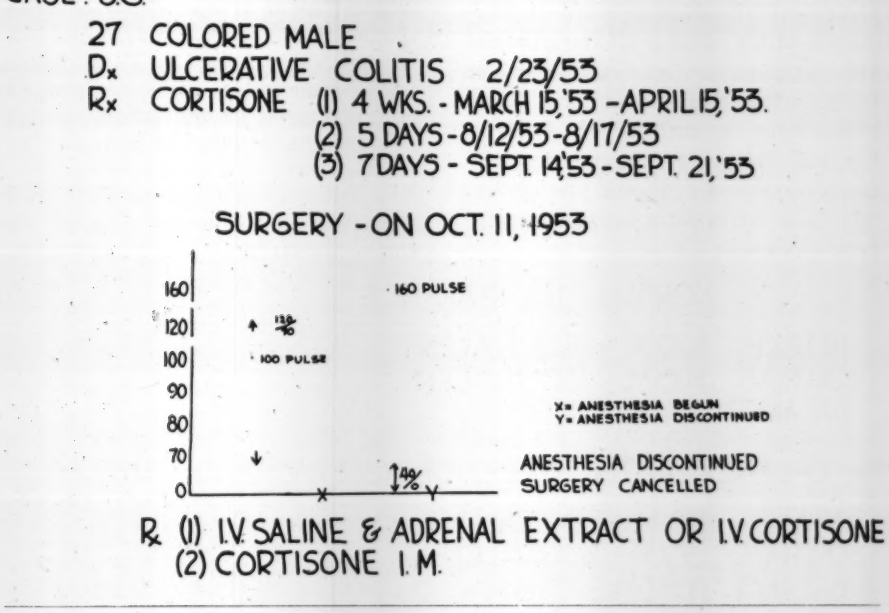

Figure 4

clinical problem that must be considered by the surgeon and the anesthesiologist. The clinical problem is threefold:

First: To select from among the many patients who have received cortisone those liable to develop adrenal cortical insufficiency as a result of anesthetic or surgical stress.

Answer: The answer to this first clinical problem is a difficult one at present. On the basis of present evidence it would seem safest to suppose that any patient who has received cortisone in any significant quantities within 3 months of anesthesia and surgery should be considered to have potential adrenal insufficiency and receive prophylactic treatment.

Second: The prevention of adrenal cortical insufficiency in those patients whom you consider susceptible of having potential adrenal insufficiency with stress.

Answer: Prevention consists of prophylactic treatment and should consist of 3 intramuscular injections (200 mg. doses) of cortisone at 24 hr. intervals prior to anesthesia and surgery and for 3 to 4 days subsequently.

Third: The recognition and treatment of adrenal cortical insufficiency in those cases in which clinical judgment and acumen was poor and prophylactic therapy was withheld or inadequate.

The patient may show evidences of adrenal insufficiency at induction of anesthesia (as in the case illustrated), during the course of surgery or in the immediate postoperative period. The first and possibly only evidence of acute adrenal insufficiency is otherwise unexplainable cardiovascular collapse with shock, tachycardia, pallor, etc.

The treatment consists of: (1) Replacement of adrenal cortical hormones which consists of the immediate intravenous administration of hydrocortisone or, if this is not available, large quantities of Aqueous Cortical Extract. Subsequently desoxycorticosterone should be given. (2) The administration of electrolytes—Na and Cl. So that it is most essential that saline be given along with cortical hormones to correct this initial unregulated Na, Cl and water loss. (3) Vasopressors are of some value—neosynephrine or norepinephrine in the saline.

Management consists then of: (1) Administration of cortical steroids—I.V. hydrocortisone preferably or if not available, aqueous adrenal extract. (2) Saline. (3) Vasopressors.

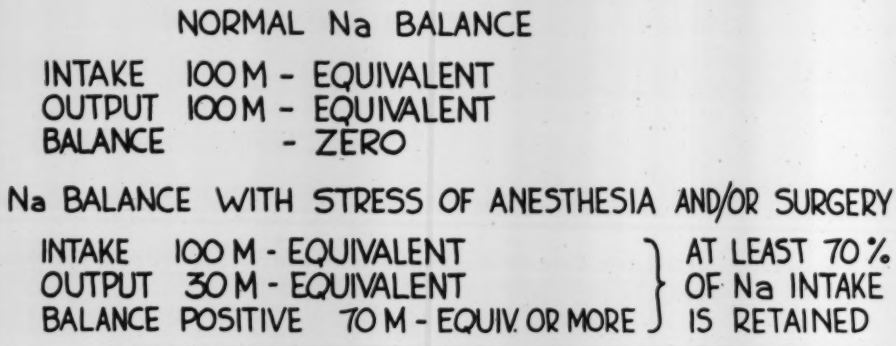

Figure 5

Postoperatively there is Na retention. (Figure 5) Under every day circumstances if the intake of Na is 100 m-equiv., output is 100 m-equiv. and the balance is zero. With the stress of anesthesia and surgery on a Na intake of 100 m-equiv., the output is only 30 m-equiv. and 70 m-equiv. is retained. So that to the usual patient undergoing surgery, saline administration is carefully restricted. However in the cortisone induced case of adrenal insufficiency, due to deficiency of cortical steroids there is an unregulated Na, Cl, and water loss with low serum Na and Cl. If the patient does not die immediately in cardiovascular collapse, the patient will die from the uncorrected disturbance of electrolyte metabolism which will produce dehydration, hemoconcentration, fall in blood pressure, oliguria and collapse.

Summary

Briefly, let me summarize. We have considered the present theoretical concept of the pituitary-adrenal interrelationship in the normal state and with stress.

Of practical clinical importance is the fact that this relationship is altered by the exogenous administration of cortisone so that as a result you may get adrenal atrophy and insufficiency. That with the stress of anesthesia and surgery adrenal response may be inadequate and you may get collapse, shock and death. In view of the ever increasing number of individuals who are and will be receiving cortisone and may have potential adrenal insufficiency it is important that anesthesiologists and surgeons be aware of the dangers and be prepared to handle any emergency situation that may arise in this regard.

An Investigation of Carbon Dioxide Retention

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The skill of anesthetists has increased so steadily that it is safe to predict that soon anoxia will never complicate well-planned operations on patients with normal cardiopulmonary systems. The anesthetist's full knowledge of the pharmacologic properties of agents employed and her meticulous attention to details related to the tracheobronchial system have brought this goal within easy reach. Efficient elimination of carbon dioxide is another objective nearly as important as anesthesia without anoxia. However, the removal of carbon dioxide during anesthesia has proved in many ways to be more difficult than the maintenance of satisfactory oxygenation. More than thirty years have elapsed since the initial studies of this problem, and yet it is certain that the goal of anesthesia without carbon dioxide retention lies some distance ahead.[1]

The clinical state of respiratory acidosis, the effect of faulty elimination of carbon dioxide, does not stand as a clear picture in the minds of most physicians. Proof of this can be found in many current surgical journals; the reasons indicate a defect in medical teaching which is outside the scope of this paper. This generalization may not apply to nurse anesthetists but a review of the effects of excess carbon dioxide may improve the understanding of some signs manifested by many patients who undergo thoracic surgery. It is the purpose of this paper, therefore, first to summarize pertinent studies in physiology. Following this, the results will be given from measurements on patients who underwent thoracic surgery on the Surgical Service of the College of Medicine of the University of Cincinnati. Finally, a limited number of inferences will be given along with some tenuous conclusions.

In summarizing known effects of excessive amounts of carbon dioxide on animals and man it is necessary to speak of organs or systems individually and perhaps imply that their responses will be observed in clinical practice. This method of study of separate units of the body, however, must not obscure the fact that what is observed in the induction room or the operating room is a complex product formed by the interrelations of all the systems of the patient. This concept must modify the application to the patient of the rather isolated facts that follow.

The subject of the effect of vagus stimulation upon the heart has been raised many times during discussions

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of arrhythmias observed during anesthesia, and especially cardiac arrest and ventricular fibrillation. It is practically impossible to identify certainly a small number of clear-cut disturbances of physiology which account for the deaths that occur while the patient is under anesthesia. This search for an explanation of operating room and recovery room deaths has led to re-examination of the function of the vagus nerve.[2,3,4] The following conclusions are drawn from experiments on dogs, and therefore there must be caution in their application to clinical problems. Vagus stimulation has a more pronounced effect upon the heart, will slow it more if there has been a fall in pH produced by CO_2 retention. In contrast with this is the effect of anoxia which lessens the effect of vagal stimulation upon the heart. Thus, if the clinical situation were one of a "pure" physiology experiment, the patient with a slow pulse rate during a mediastinal dissection should be suspected of having respiratory acidosis, whereas in a comparable situation if the pulse rate speeded the patient should be considered anoxic. If both hypercapnia and hypoxia are present, the effects of hypoxia dominate the clinical signs and the vagal tone diminishes, accounting for a rapid pulse. It is worth restating, finally, that no investigator has produced cardiac arrest or ventricular fibrillation by the single mechanism of vagal stimulation.

In the state of hypercapnia it is now known that the excessive concentrations of carbon dioxide in the plasma have direct effects upon the heart. Laboratory experiments have shown, first, that the muscle elements are unable to contract with normal force if an excess of CO_2 is present; with this effect fully developed, there is a marked cardiac dilatation.[5] Serious changes in the electrocardiogram have been recorded in patients who breathed carbon dioxide, and it is possible that these illustrate the known direct effect of CO_2 on the animal heart.[6] Conduction of the impulse through the auricle (A-V conduction) is depressed when carbon dioxide has accumulated, and finally complete heart block may result. In addition, there is depression of the special tissue (SA node) which starts the impulse into the ventricular walls, and associated with this may be irregular rhythms.[7]

With these details it is pertinent to comment upon cyclopropane and the hazards some attribute to its use. It is the authors' opinion that all the untoward effects upon the heart and vascular system are attributable to the hypercapnia which accompanies cyclopropane administered without perfect ventilation. We believe that cyclopropane uncomplicated by hypercapnia has not been proved to be the cause of arrhythmias.[8]

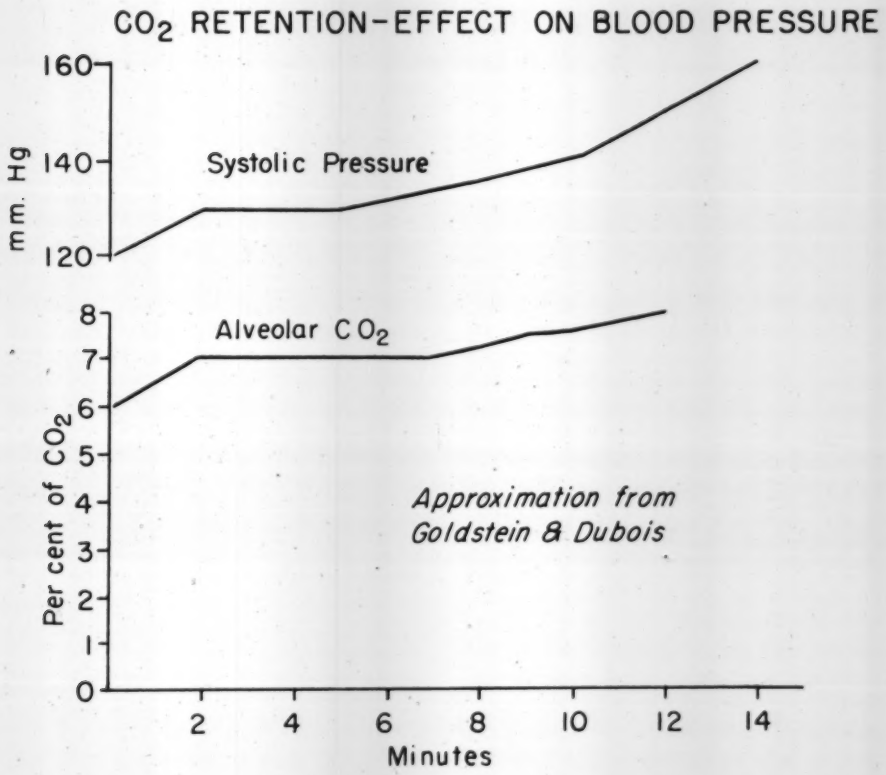
The effects upon the central nervous system of acidosis caused by carbon dioxide retention can be outlined in the following three parts. First, CO_2 has definite narcotic action.[9] Consciousness is lost if carbon dioxide is present in the alveolar air in concentration of 11% or more. In addition, this gas is known to have summative narcotic action when present during anesthesia maintained by pentobarbital. The duration of pentobarbital anesthesia is significantly lengthened if acidosis develops due to improper elimination of carbon dioxide. It seems to us that this point has received much less attention than it deserves in the daily practice of anesthesiology.

The second central effect of CO_2 is its stimulation of the respiratory

center. A rise of only 1.6 mm. Hg. in the partial pressure of carbon dioxide in the alveolar air will double the ventilation as the result of this potent stimulant effect upon the respiratory center via the arterial blood.[10] Pulmonary ventilation will increase as the concentration of CO_2 in inspired air rises until approximately 9% is reached. Above that level the stimulant effect diminishes and, as mentioned, narcosis develops.[11] Under many anesthetics the strength of respiratory stimulation from CO_2 is diminished and this may be the very basis of the respiratory acidosis problem: Our anesthetic agents dull the patient's physiologic responses and carbon dioxide may accumulate without exciting an increase in ventilation which would eliminate this metabolic product.

The third effect of CO_2 upon the central nervous system is concerned with the vasomotor center. Hypercapnia as a cause of elevation of the blood pressure is an axiom which needs little further discussion. Several ancillary points, however, deserve emphasis, and this can be done best by reviewing the results of Goldstein and DuBois.[12] The systolic pressure rises in at least two steps and the initial rise in diastolic pressure is slower. (Fig. 1) In the recovery period, or as the patient rapidly eliminates CO_2, the systolic pressure falls to normal in 4 to 6 minutes. The

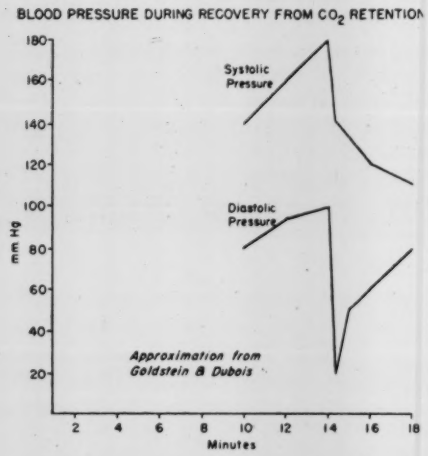
Figure 1



diastolic pressure falls abruptly below normal and then quickly rises to nearly normal levels (Fig. 2).

Additional information related to serum potassium levels has some bearing on the interpretation of these observations on blood pressure. As respiratory acidosis develops and the pH falls below normal, the concentration of potassium increases in the serum.[4] This elevation of serum potassium continues to still higher levels

Figure 2



in the first few minutes *after* the experimental animal starts rapidly to eliminate carbon dioxide. Animals in severe acidosis regularly develop ventricular fibrillation within a few minutes after starting to breathe air (instead of a high concentration of CO_2).[13] Thus the highest level of serum potassium is found shortly after the alveolar CO_2 concentration starts to diminish and it is at this juncture that the experimental animal fibrillates and dies. Every anesthetist and every surgeon can recall a patient who manifested some of the signs of respiratory acidosis, and weathered a difficult operative procedure only to succumb abruptly as he was detached from the anesthetic apparatus and started to breathe air. The facts now available relative to cardiovascular response and alterations in serum potassium in association with CO_2 retention permit a clearer analysis of operating room deaths. We urge all anesthetists to analyze critically the records of any of their patients who fall in this group and to interpret them in the light of the above facts.

The facts given above would not satisfy a physiologist requesting a full discussion of the effects of excessive concentrations of carbon dioxide. We have omitted mention of such details as the local action of CO_2 on the coronary arteries, other veins and capillaries, as well as the chemoreceptors of the carotid sinus and aortic arch. However, the outline given covers the most important reactions and certainly the ones anesthetists and surgeons must understand for the safety of their patients.

Description of Anesthetic Technique

Anesthesia was induced in every case with a small dose of thiopental sodium. It was administered slowly and in an amount that barely ablated the lid reflex. Enough ether was then given by open drop to carry the patient into the third plane so that the glottic opening could be exposed and the trachea intubated. All operations were performed with the patients in the lateral recumbent position. A to-and-fro system was employed and this included an exhalation valve adjusted to permit free escape of oxygen which was added in amounts that exceeded the patient's requirements. The standard adult size cannister of soda line was used during all operations except those on children less than three years old for whom the infant size was employed.

The administration of ether was continued in many cases while in others cyclopropane was the agent for maintenance. The anesthetist gained complete control of respirations a short time before the pleural space was opened and she continued with this ventilation technique until the musculature of the chest wall was sutured. Meperidine was frequently used as a supplementary agent. "Packing" or compression of the lung by the surgeon was always planned to avoid excessive encroachment and still provide satisfactory exposure.

The technique employed for the operations on adults was essentially the same with the exception of two details. A topical anesthetic was applied by transtracheal injection and the gases were administered in a circle system.

Results

The first group of patients to be summarized are those who had resection of the thoracic aorta for coarctation. Since there were only four, the averages given are of limited value and do not permit broad conclusions. It will be seen later, however, that there is an interesting comparison between these results and the ones obtained from larger groups of patients with other diagnosis. Blood sample number one was drawn immediately after induction of anes-

Figure 3

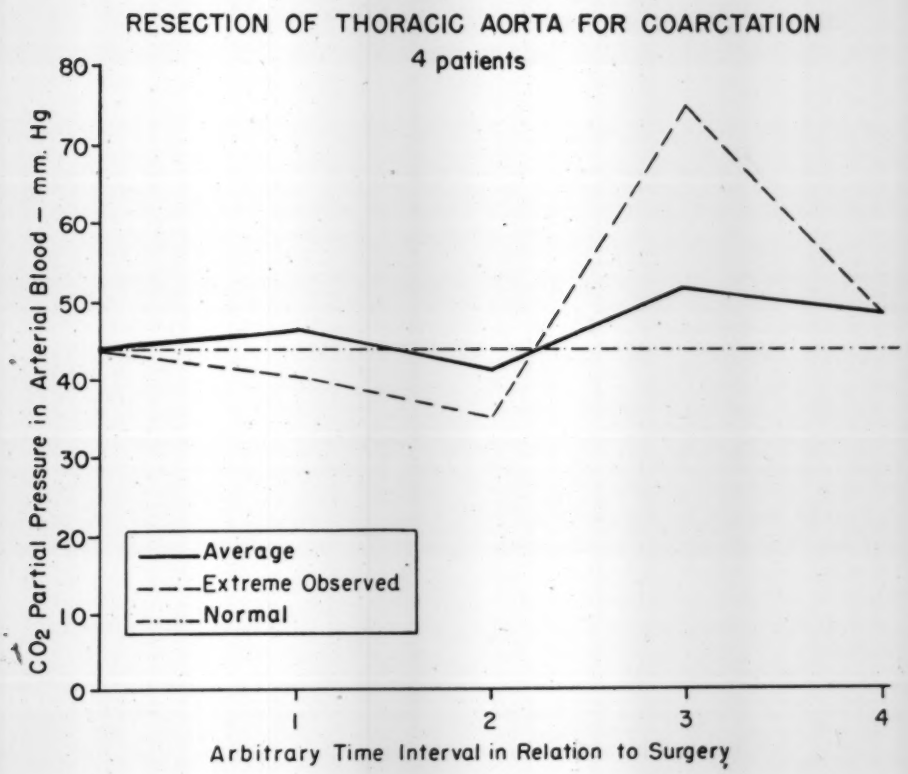
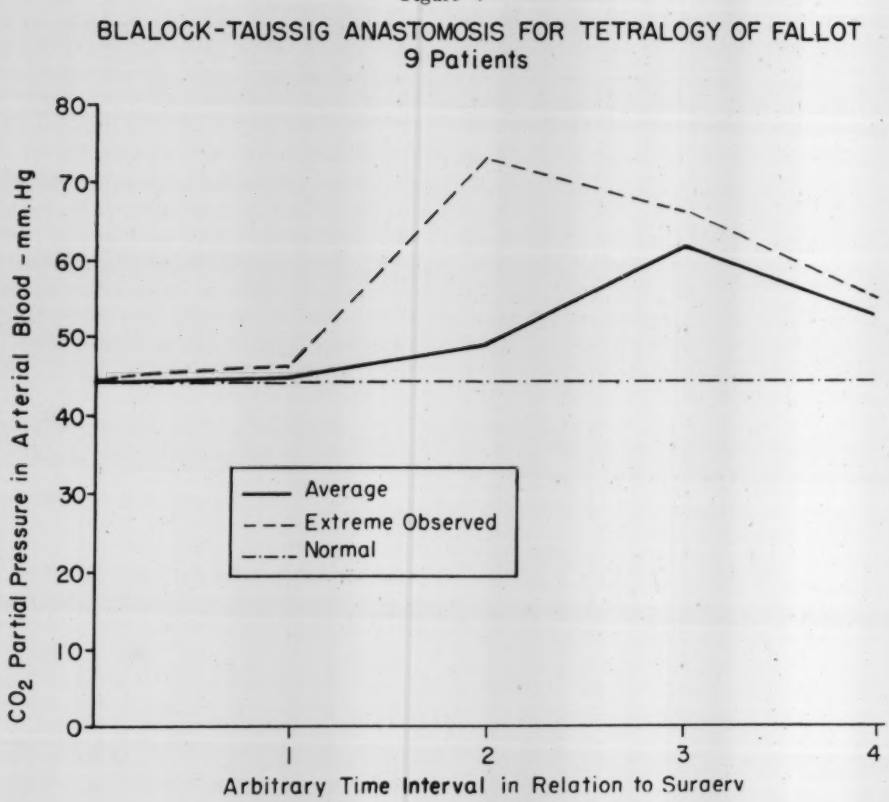


Figure 4



thesia, number two as soon as the pleural space was opened, number three at the conclusion of the intrathoracic portion of the operation, and number four after the wound was closed and the patient turned onto his back. (Fig. 3) Perhaps the most significant finding was the extreme acidosis observed in one patient at the end of the aortic anastomosis. The possibility of this complication should be kept in mind when a patient's condition deteriorates at the completion of a difficult procedure. Trauma and blood loss may be contributory but respiratory acidosis may be of equal importance and yet be entirely unrecognized by the unwary.

The next group of patients were nine children with tetralogy of Fallot. They were not critically ill but certainly there was serious disturbance of the circulation through the lungs with an unknown effect upon the exchange of CO_2 during the anesthesia. A moderate degree of respiratory acidosis was the average result from blood samples drawn after completion of the Blalock-Taussig anastomosis (Fig. 4). Considering the entire course under anesthesia, however, the removal of CO_2 was quite satisfactory and this confirmed our clinical impression of the patients.

The third group was comprised of five adults with mitral stenosis. This cardiac abnormality also seriously

affects the pulmonary circulation and therefore may disturb the exchange of CO_2 across the respiratory surface. Even without an understanding of this possible alteration in lung function, it is of interest to review the results obtained on analysis of the samples of arterial blood (Fig. 5).

Note in the first place that the degree of respiratory acidosis was greater at the time of the first blood sample, i.e., immediately following induction of anesthesia, than in any of the three samples drawn subsequently. It was found also that the average of CO_2 tensions indicated only a slight degree of acidosis at the conclusion of cardiac manipulation and at the termination of surgery. This suggests that despite the serious cardiopulmonary disability of the group, the proper use of the anesthetic technique described will give acceptable results as judged by carbon dioxide elimination.

The fourth group includes twelve children with patent ductus arteriosus. These patients were in excellent condition, though again, it is mentioned that their pulmonary circulation was abnormal, and this in itself may have influenced the elimination of carbon dioxide. It should be noted first that a moderate degree of acidosis was present at the time samples number two were drawn (Fig. 6). This, of course, was just after the pleural space had been opened so

Figure 5

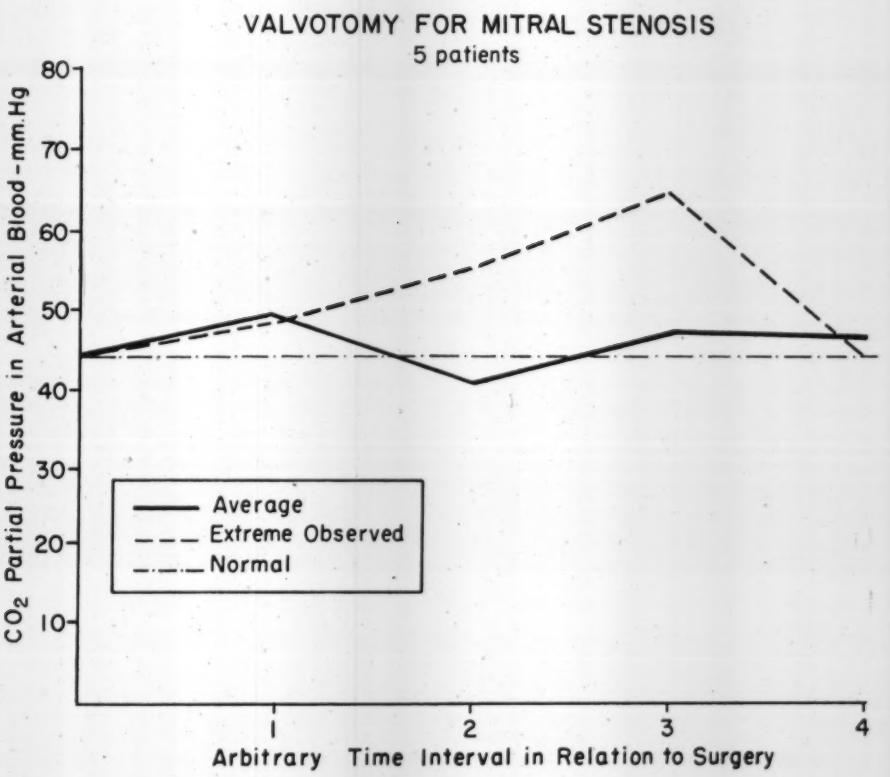
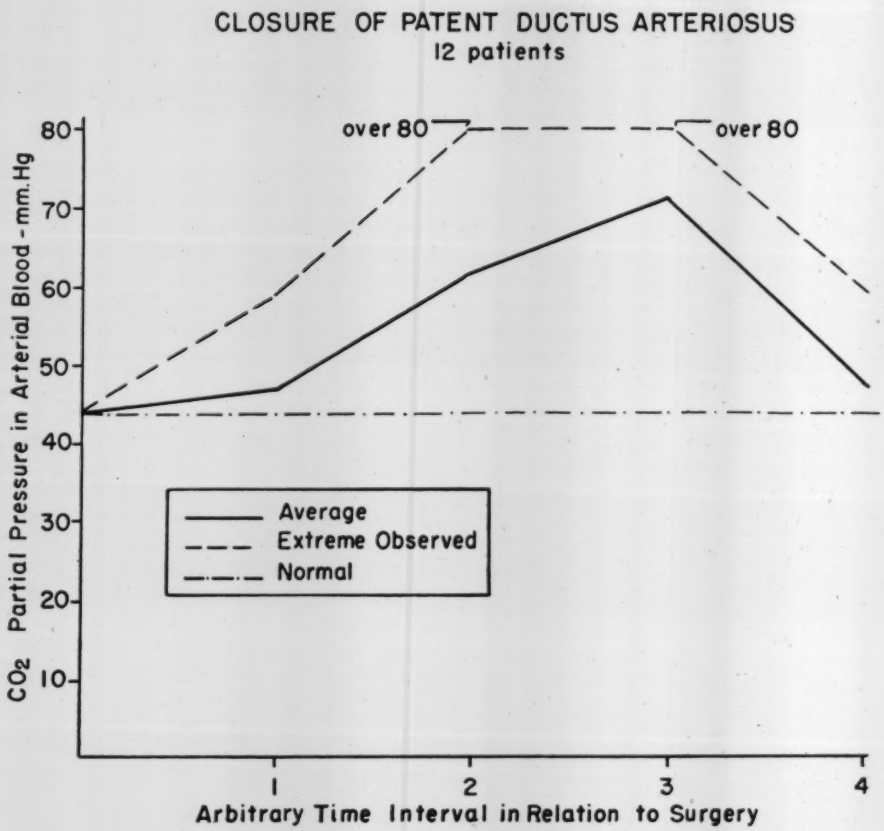


Figure 6



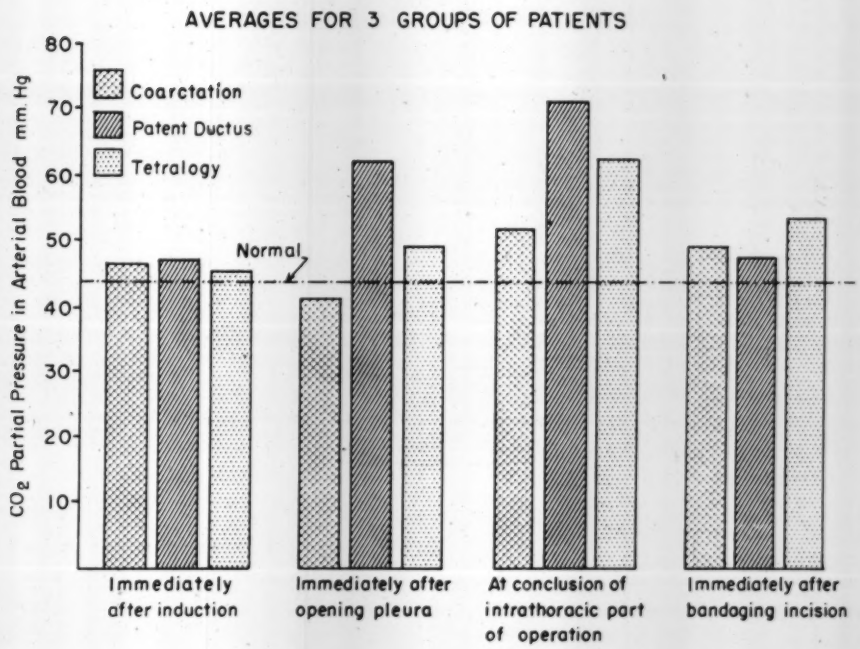
that, aside from the fact that the patients had been lying in the right lateral position, no unusual conditions obtained that would affect pulmonary ventilation. By the time the ductus had been divided and sutured the acidosis had progressed still further. There was improvement in CO_2 elimination as the chest wall was closed, however, and on the average, the sample at the end of the operation indicated very little CO_2 retention. The severest respiratory acidosis encountered was in a patient in this series, and this emphasizes one of its aspects as an anesthetic complication with a known effect upon morbidity and mortality rates. The well standardized operative procedure for ductus arteriosus has a risk which must be practically nil. The anesthetist and the surgeon must be doubly vigilant, therefore, to prevent any harmful effect from acidosis in children who now can be cured without risk inherent in the surgical technique.

The next table restates the averages of CO_2 tension for each of the four periods at which samples of arterial blood were drawn and for the three groups of children, omitting the adults with mitral stenosis (Fig. 7). Despite the great differences in

cardiopulmonary physiology which must separate coarctation, patent ductus and tetralogy of Fallot, each group developed mild hypercapnia immediately following induction. Factors responsible for this change must operate with the same force in each different group at this stage of the anesthetic period. After a period of lateral recumbency and within a very short time after opening the pleural space the average of results indicated a moderately severe degree of CO_2 retention in the patent ductus group. We doubt that the results obtained for coarctation and tetralogy groups indicate a significant change. The period of intrathoracic manipulation was one in which all the children became more acidotic. The average for those with coarctation showed the least change, the change was intermediate in children with tetralogy, while the patent ductus group averaged a CO_2 tension indicative of a serious state of respiratory acidosis. Factors responsible for improved elimination of carbon dioxide had effect upon all three groups so that at the conclusion of surgery the averages of CO_2 tensions were approximately the same respectively as very early in the anesthetic period.

An additional statement may be required to place the subject of carbon dioxide retention in proper perspective. It is our belief that the degree of acidosis experienced by these patients causes a reversible physiologic response. There were no deaths in these groups, nor were we able to prove that the hypercapnia increased the morbidity. But acidosis of the degree encountered certainly adds to the burden of impaired function that must be borne by the patient. This

Figure 7



addition may be intolerable if there is associated with the operation the additional stress of uncompensated blood loss, excessive trauma, or anoxia. Hypercapnia added to any combination of these other unphysiologic conditions may be lethal and indeed we suspect that it accounts for some operating room deaths which formerly could not be explained.

Summary

We have presented a review of the disturbance of function that is caused by retention of carbon dioxide. This does not cover all the physiologic responses to hypercapnia, but it lists reactions which much be understood by anesthetist and surgeon. Thirty patients, 25 children and 5 adults, were studied for evidence of carbon dioxide retention under anesthesia for cardiovascular surgery. They were grouped according to diagnosis, but all showed a comparable degree of respiratory acidosis immediately following induction. All groups manifested a greater degree of CO_2 retention at the conclusion of the intrathoracic stage than at the beginning of the intrapleural surgery. Patients with patent ductus arteriosus developed the severest acidosis, and for this reason present a special problem for anesthetist and surgeon. The importance of hypercapnia as a complication of anesthesia for cardiovascular surgery has been reaffirmed and the frequency of its occurrence in children is established by the data in this study.

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The Nursing Profession and A Quarter-Century of Progress in the Relief of Pain and Distress

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In the work of some anthropologists can be found evidence that the first anesthetist could be said to be a woman. This woman was the head of the primitive family, and she was known as the "Great Mother." She was a priestess and sorceress, and such was the nature of her ministrations that she might be thought of as the founder of the healing art. When a primitive sick man could not relieve his own suffering, he called upon the priestess to banish his pain. Exactly how she did it is still a mystery, but conjurations and spells undoubtedly were used and magic was applied, and perhaps in some cases pain was thereby abolished.

There is little or no mystery about present-day anesthesia. It is still true that we do not understand the basic mechanisms responsible for the control of pain, but control and alleviation of pain in most instances can be achieved by knowledge of drugs and by skill in the use of them. It is not too much to say that the progress made in this field in the past 25 years is as remarkable as, and certainly more dramatic from the standpoint of conservation of life than, the primary discovery of abolition of pain itself during surgical operations.

The reason for this is the great variety of agents and technics now available. These have been introduced and applied during the last 25 years. I am concerned here, as you are, only with those persons who are under professional treatment. With that little group of wilful souls who have real or imagined complaints which they treat themselves we have no concern, as they have none for us. I shall, then, direct my remarks very largely to measures used in dealing with operative pain, but I shall also include a few remarks on distress of other kinds.

Consider, first, a few of the major agents currently at hand. We have, by this time, amassed a considerable experience with diethyl ether and nitrous oxide and oxygen, and ethylene. In the early days a great deal was done with chloroform, an agent which for one reason or another could not retain favor. As the variety of volatile anesthetics and gaseous agents increased, attempts were made to reduce the doses, so that more oxygen could be administered with them. This objective in turn led to an attempt to utilize preliminary medicaments in various doses, plus a local anesthetic agent, especially in the infiltrating of the line of incision.

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Balanced Anesthesia

Thirty years ago I spoke on this subject under the term, "balanced anesthesia." About that time or before, Dr. J. T. Gwathmey was speaking about the synergism of drugs, and he pointed out the synergism between magnesium sulfate and morphine when these agents are given hypodermically. In 1920 Dr. George W. Crile, who had much to do with the development of the nurse-anesthetist, contributed the technic of anoci-association, which is the combination of preliminary medication, local infiltration of an anesthetic agent and administration of a general anesthetic agent. Behind these early efforts to use multiple agents, each in a small dose, was the objective of diminishing the surgeon's reliance upon a large dose of a single agent, as when ether, given by the open-drop method, was depended upon alone to produce anesthesia and relaxation.

The giving of a large dose of a single anesthetic agent certainly produced better results than in the days before anesthesia was possible, when patients were restrained by force, without freedom from pain, yet many discouraging situations arose. A vivid example might be the use of the foot pump and the Junker bottle to introduce ether vapor into the oropharynx for maintenance of anesthesia in children during tonsillectomy and adenoidectomy. Many a time this procedure was a bloody struggle — so much so that often, when operation for cleft palate and harelip was to be done, chloroform had to be used. Yet, when the surgeon traded the blood and confusion for a quiet patient, an increased risk to life had to be accepted.

Additions to the Ether Series

One of the additions to the ether series came just about 25 years ago, when Dr. Chauncey D. Leake predicted that divinyl ether would come into use as an anesthetic agent. In the following year or two the drug was synthesized and produced in pure form. It has been available ever since. Divinyl ether produces anesthesia quickly, and is especially useful for an operation such as myringotomy and measurement of pressure within the eyeball. The recovery period is short, and few untoward results follow the use of the agent in this manner. Divinyl ether of course can be used to reinforce the effect of nitrous oxide and oxygen; it is sometimes used in this way in dental surgery.

Samuel Goldschmidt and I. S. Ravdin and associates recommended the anesthetic use of divinyl ether in 1933, the year in which the *Bulletin of the National Association of Nurse Anesthetists* began publication. In the next year I introduced pentothal sodium as an anesthetic agent for intravenous administration.

Work With Carbon Dioxide

Thus far we have dealt with anesthetic agents. The use of carbon dioxide is another matter. Henderson and Haggard had suggested that carbon dioxide might be used to compensate for the situation in which, as the depth of respiration is doubled, the rate of absorption of ether also is doubled. As a result of this, in 1923 I specified and Foregger built for me a gas machine of such design that nitrous oxide, oxygen, ethylene and carbon dioxide could be administered with it. The machine also had a vaporizer for ether. The apparatus or the idea behind it was given a variety of trials. One man, Dr. Ben Morgan, finally utilized 20 per cent carbon dioxide plus oxygen with ether to induce sudden anesthesia. This he accomplished, but the method was soon abandoned.

About this same time Dr. Ralph Waters, who had studied with Dr. Dennis Jackson at Western Reserve University, developed a method for the use of soda lime (sodium and calcium hydroxide and bara lime (barium hydroxide and calcium hydroxide) to absorb carbon dioxide in a recirculated mixture of gases. This was fortunate, for if this method had not been available, it would have been difficult 10 years later to introduce cyclopropane. Cyclopropane was so expensive that to use it only once, and to allow it to dissipate, would have been prohibitive. The work of Waters and others made it possible to use the agent over and over again, simply by the addition of oxygen to the mixture. The patient converts most of the oxygen into carbon dioxide, and the soda lime removes a large part of carbon dioxide, leaving a mixture that is predominantly cyclopropane. If there are no leaks, anesthesia can be carried on in this manner very economically.

However, as a result of the economy that had been achieved by this technic with cyclopropane, there was a gradual tendency to try to use all inhalation anesthetic agents in this manner. Before long it seemed to me that many were doing nothing more than to administer ether and oxygen by the closed method, to the exclusion of other useful and efficacious technics. Both Dr. J. A. Heidbrink and I became concerned about the fact that the art of producing anesthesia with nitrous oxide and oxygen was in danger of disappearing.

Muscle-relaxing Agents or Technics

A variety of anesthetic agents, both local and general, were tried for the next 6 or 8 years, but the most startling announcement was that of Harold Griffith, in 1942, of his successful use of curare. This news set off a chain of events which initiated an extensive search for muscle relaxants and for antidotes for them. Thus, the use of neostigmine to counteract the effect of curare was improved upon by the development of edrophonium (tensilon choloride). The result was that flexibility was added to the use of curare, and so also was a considerable factor of safety. This was most important, as we shall see.

Over the years there had been no particular difficulty in making a patient unconscious of pain, and a number of agents were available for this purpose. Nevertheless, the surgeons needed more than that. To operate successfully in certain cases a greater degree of muscular relaxation than previously had been possible was very much needed. To obtain such relaxation greater doses of the anesthetic agents were used, and many times the patient was overdosed. An increase in postoperative complications and even deaths was the inevitable result. Hence, one of the great virtues of spinal anesthesia is the complete muscular relaxation which it produces. However, in the hands of some persons spinal anesthesia gave untoward results, so that when it became possible to achieve adequate muscular relaxation well and quickly with curare, the use of spinal anesthesia declined. As a matter of fact, the use of spinal anesthesia may have declined too much, because there are occasions when it is still the method of choice.

The development and introduction of continuous spinal anesthesia by Lemmon[5] in 1940 increased the utility of spinal anesthesia, but the method required mastery of a special technic and the use of special equipment. Since the tendency to follow the course of least resistance applies in anesthesia as well as in other en-

deavors, the technic of continuous spinal anesthesia lost favor. I am not sure that this is good.

It is nonetheless true that the development of succinylcholine for the production of muscular relaxation satisfied an important need. The special advantage of succinylcholine is that it will bring about relaxation of the musculature of the throat and the larynx. On the other hand, no antagonist has yet been developed to counteract the effects of succinylcholine, so that the use of the agent usually is confined to facilitation of intubation to maintain an airway when pentothal sodium anesthesia also is to be employed. The combination of pentothal sodium and curare does not eliminate laryngeal spasm. This means that to date a better intravenous anesthetic agent than pentothal sodium has not been developed. Succinylcholine serves a very useful purpose in that once the intratracheal tube is in place, curare can be administered.

The Problem of Anoxia

Today, then, probably the most generally used combination of anesthetic agents is pentothal sodium and succinylcholine for intubation, followed by additional pentothal sodium and curare. Oxygen and nitrous oxide are given by intratracheal tube. I am trying not to say *nitrous oxide* and oxygen, but rather, to emphasize the need for *oxygen*. I stress *oxygen* for a very sound reason. As surgeons began to attempt operations which became more and more extensive, the thorax as well as the nervous system, the skeleton and all the viscera naturally came to be involved. There were days when the anesthetist did not have recourse to the many drugs now available, so that on occasion respiration was unduly suppressed and anoxia developed. We then began to hear a great deal about "cardiac arrest." It is my feeling that anoxia is a very important contributing factor to cardiac arrest, and that good oxygenation should be maintained at all times. Anoxia, even when present for periods as short as 2 or 3 minutes, may cause trouble.

It has been said that the use of curare increases the mortality rate over that among anesthetized patients who do not receive curare. My experience is contrary to this view. I believe that if the mortality rate was increased by the use of curare, investigation would show that it could not be charged so much to the drug as to the way in which the drug was used. Very likely it would be found that respiratory depression from the combination of pentothal sodium and curare resulted in anoxia, and that the difficulties encountered were due to the anoxia. If so, the anoxia could have been overcome by the administration of oxygen. We must never lose sight of the fact that the success of most agents used by the anesthetist depends upon the skill with which they are used, and that a valuable agent actually is not good when it is separated from the skill of the anesthetist.

Two New Intravenous Agents

Two new intravenous anesthetic agents are being investigated. One is dolitrone and the other is viadril, but the occurrence of thrombophlebitis in a certain percentage of patients when these drugs are used intravenously has retarded the use of both of these agents. However, I think we have learned a great deal from them. When dolitrone is used in full dose an anesthetic effect is obtained; when it is used in half dose an analgesic effect is produced; when it is administered in one-quarter dose, amnesia is effected. No doubt there is a place for such effects, and if dolitrone and via-

dril cannot be made to produce them without complications, attempts will be made to find other means to generate them.

Tranquilizing Agents

It seemed to me that the recently introduced tranquilizing drugs, such as promethazine hydrochloride, (phenergan hydrochloride), chlorpromazine hydrochloride (thorazine hydrochloride), equanil and so on would be useful in preliminary medication. I have now used such agents in preliminary medication long enough to be convinced of their importance. As an example, I try to give an adult patient 25 mg. of promethazine hydrochloride (phenergan hydrocloride) by mouth at bedtime, together with 500 mg. or less of placidyl, to enhance the possibility of a good night's sleep. However, when I call upon the patient the next morning and find that he has not slept well, I know he will be resistant to drugs. Conversely, I know that the patient who has slept well will not need large doses of drugs. Hence, in the morning (a half-hour or more before operation) I will give the patient 25 mg. of phenergan hydrochloride intramuscularly plus 2 mg. of levorphan tartrate (levo-dromoran tartrate) and 1/150 grain of atropine. Anesthesia for these patients then can be instituted and maintained with much smaller doses of pentothal sodium than is usual. In some cases the doses of pentothal sodium have been reduced by 50 per cent, and in others by a third to a fourth of the former dose.

The advantages of such a technic are particularly noticeable in prolonged operations such as extensive vein stripping. Phenergan hydrochloride markedly reduces nausea in most people, and I have used it to advantage for patients who are to undergo hemorrhoidectomy under sacral block anesthesia. Many of these patients previously used to become nauseated and to vomit, but after the use of phenergan hydrochloride was started, only about 1 in 100 patients became nauseated. We particularly noticed the change because it used to be necessary to have oxygen on hand for administration to these patients, and now oxygen is seldom used. In the postoperative period, because of the synergistic effect which phenergan hydrochloride exerts on certain drugs such as levo-dromoran, we have been able to relieve the pain that follows hemorrhoidectomy. The achievement of such relief is striking to all concerned. Without a doubt, we now have the most effective control of such pain that was ever possible.

Other drugs have been tried for sleep and in proper doses they give satisfactory results. For example, with phenergan hydrochloride it is possible to use methyprylon (noludar), glutethimide (doriden) or pentobarbital sodium (nembutal) or similar drugs. Thorazine can be used instead of phenergan hydrochloride, although for some reason I have been so well pleased with phenergan hydrochloride that I have continued to use it. On the other hand, a tranquilizing drugs such as equanil does not exert a synergistic effect on other drugs. This means that if it were to be used, exceptional caution would not have to be observed in the matter of how much morphine, levo-dromoran, anillerdine or similar agents should be given, should the concomitant use of any of them be desirable.

By contrast, it is important to remember that if phenergan hydrochloride is used both at night and in the morning, the dose of morphine should be a little smaller than usual. If the patient is robust, it probably

would be better, after he has had phenergan hydrochloride, to administer 1/6 grain of morphine rather than 1/4 grain, and if the patient is of average size, the dose might be 1/8 grain. Futhermore, after the patient has received medication such as I have described, the person administering the anesthetic agent must be guarded in the matter of the rate at which he gives pentothal sodium, and the dose of the agent must be small. He must also give ample oxygen. For example, if the flow of gases is 8 liters of nitrous oxide and 2 liters of oxygen per minute, the arterial oxygen saturation probably will decrease to about 90 per cent. If, on the other hand, 7 liters of nitrous oxide and 3 liters of oxygen per minute are allowed to flow into the breathing bag, the arterial oxygen saturation probably will be approximately 100 per cent.

Complications of Surgical Operations

Many other situations arise and must be managed summarily. During certain operations, for instance, nausea and vomiting can be great hazards. If they occur, 50 mg. of cyclizine lactate (marezine lactate) can be given intramuscularly. This agent is usually very effective and rather quick in its action. Nausea is very distressing to the patient and the surgeon, and it is a contingency that must be kept in mind constantly.

Chronic Pain

In the relief of pain, and particularly chronic pain, circumstances arise which often lead to overdoses of pain-relieving drugs. The agent that comes to mind first in this respect is morphine, because it has been the drug most widely used for the relief of pain. In these days of a large population and a considerable number of persons with carcinoma, the problem of terminal cancer presents itself, in which severe pain must be dealt with for a prolonged period. So far as morphine is concerned, we are now fortunate in the fact that a new product, nalorphine hydrochloride (nalline hydrochloride) will very quickly neutralize the effect of morphine, particularly in the matter of respiratory depression.

A note of caution is required when antidotes for drugs are mentioned. The hope should not be entertained that when these antidotes are used, the patient immediately will become perfectly conscious and be able to take care of himself in every way. Actually, in the case of overdose, the factor we are more concerned about than almost anything else is respiratory depression. That is, drugs which will neutralize the effects of other drugs sufficiently to permit the patient to breathe adequately are what I mean by "antidotes" in this particular context. Speedy relief of respiratory depression is the prime consideration here.

Nalline hydrochloride was introduced with impressive recommendations, and it still serves a useful purpose. It is, however, expensive. It has the peculiar property that if the drug that produced the overdose is unknown and nalline hydrochloride is administered to neutralize the overdose, respiratory depression will be increased, rather than relieved, if the overdose was caused by a barbiturate. This property in fact might be regarded as a diagnostic aid in inverse character, since it might help by exclusion to establish the knowledge that the drug the patient had taken was not morphine. Curiously, until very recently, we had nothing that would antagonize the effects of nalline hydrochloride except morphine. Few of us would be bold enough to ad-

minister morphine for such a purpose.

Recently an agent called "lorfan tartrate" has been developed. It exerts an antagonizing effect on narcotic agents without producing undesirable side ecects, so far as I know, and it is not too expensive. Another drug which is said to relieve the respiratory depression caused by a barbiturate is *B, B*-methylethyl glutarimide (megimide).[6,7] Daptazole (amiphenazole) is a new agent that is said to be able to antagonize the effect of morphine, especially in the matter of respiratory depression.

Problems Peculiar to Certain Drugs

The number of drugs available today is increasing rapidly, and I foresee a time when most situations, very difficult in the past, can be managed with satisfaction. However, we must remain constantly on guard to ensure that the introduction of so many new agents does not create artificial hazards. An example is afforded in the case of cortisone. It was found, after a while, that patients who had been receiving cortisone for a long time and then ceased to receive it were almost certain to have atrophy of the adrenal cortex. The result was that they became quite unable to withstand the stress and strain of a surgical operation, and if they underwent such a procedure they would go into irreversible shock and die. Now we know that such patients can be prepared for surgical operations by the administration of 100 mg. of cortisone intramuscularly each day for 2 or 3 days before the operation, on the day of operation and for 2 or 3 days after operation.

Hydrocortisone (compound F) has been made available in lyophilized form, solu-cortef, so that an intravenous injection of 100 mg. of the drug in as little as 2 cc. of sterile distilled water will, as a rule, provide the patient with considerable support. On rare occasions this may not be enough, and it may be necessary to use some vasopressor agent, or even adrenocortical extract. However, such a situation would be rare. Neurogenic shock can occur when a patient has been given preliminary medication such as I have mentioned, and then has been operated upon in the upright position under local anesthesia—perhaps tonsillectomy or operations on the nose. Such a patient may become cold, wet and white; nausea and vomiting may ensue; the blood pressure and the pulse pressure are low. But even in such a case most patients will respond to the intravenous administration of 25 mg. of ephedrine to increase the blood pressure. It is true, however, that some of them do not feel well again, even if the blood pressure does rise. If that proves to be true, the patient usually can be made to feel well by the intravenous administration of hydrocortisone.

When phenergan hydrochloride began to be used as a preliminary medication, it was soon suspected that this new agent increased the frequency with which neurogenic shock developed in certain patients. To combat this suggestive effect, we began to administer 30 mg. of mephentermine sulfate (wyamine) intramuscularly at the time the preliminary medication was given. We judged that this generally was effective, since we did not observe many patients who had received it who were unable to co-operate with the surgeon.

Recently we have alternated wyamine and ritalin, and we think we get satisfactory results from each of these agents. There has been an increasing tendency to accept the concept that certain drugs used to produce a given effect in a given condition may cause some undesirable side effects when used against some other

condition. For example, Rauwolfia seems to be satisfactory in the treatment of certain mentally disturbed persons, but when it is used for the treatment of hypertension it may overly depress the patient. The result is that ritalin now is embodied in the same dose with the Rauwolfia, and the ritalin is supposed to eliminate the undesirable degree of depression in the hypertensive person caused by the former agent. At first it was somewhat difficult to grasp the basis on which this procedure was based, but it seems to be well established now. It indicates that in the future we no doubt will have to reason quite differently in such matters than we did in the past.

Certain patients who suffer greatly from, say, terminal carcinoma, complain that they cannot take morphine without experiencing nausea. It is possible in these instances to offset the effects of morphine by giving a dose of hyatrobal 30 minutes before the morphine is to be given. This usually will control the nausea. For some people dramamine is effective. It is useful to keep in mind two or three drugs that will produce a given effect in most people, so that if a certain patient does not respond to one of the drugs, another may be administered. There is much to be said in favor of an extensive knowledge of the many drugs now currently available, even though the great diversity of them at times seems overwhelming.

Block Procedures

While nurses are not called upon to make diagnostic and therapeutic blocks, still it is important for them to know that such procedures can be used and that they are very helpful. For example, alcohol block of the stellate ganglion has been used to relieve the pain of angina pectoris, and atypical facial pain, and the pain of the arm-hand syndrome, in which the hand has been injured and the arm becomes swollen, painful and useless. Physiotherapy cannot be inaugurated or carried out until the block has been done. Lumbar chemical sympathectomy with alcohol or even with a 6 per cent solution of phenol and water has been very effective for patients who are poor operative risks and even for some who are not. Sometimes the block has been effective for as long as a year. If the pain pathway can be blocked, it often helps in differential diagnosis of the patient's condition. Two factors are important in alcohol chemical sympathectomy: (1) roentgenograms should be taken to show where the needles are, and (2) sweating tests should be conducted afterward, to determine whether the block actually did destroy the fibers and thereby prevent sweating in the area being treated. A mixture of 2.5 per cent ammonium sulfate, 2 per cent benzyl alcohol, and 1 or 2 per cent procaine hydrochloride (called "PAB") can be used for diagnostic block without the need for roentgenograms. This mixture also may be used for the temporary relief of pain. In patients who have thoracic injuries it may be used for intercostal blocks, to allow easier breathing and to decrease pulmonary edema.

Comment

Those who are active in the care of patients today should especially appreciate the great variety of agents and technics at hand which were not available 25 years ago. Concomitantly, the modern nurse has to know more now than the nurse of 25 years ago knew. The history of anesthesia,[8,9] embracing some 110 years, actually is a notable success story, for 100 years is not much more than the

life span of a long-lived person. It may seem a long time to us, but in relation to medicine the period actually is hardly more than the flutter of an eyelash. The success which we enjoy now was no accident; it was brought about by the expenditure of much effort, research and clinical investigation. Like most objectives of genuine value, it was achieved by the exercise of boundless energy by an endless number of people. If the progress made in the last 25 years is any indication of a marked increase in the tempo of development of better and better drugs and equipment, then the next 25 years probably will be characterized by one success after another. The ultimate achievement cannot fail to be vast improvement in the efficacy of our labors.

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Obstetric Anesthesia
Some Prejudices and Convictions

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At this twenty-third annual convention of the American Association of Nurse Anesthetists many of the major problems of anesthesia have been ably and excellently discussed. Obstetric anesthesia, perhaps a lesser field to many of you, has long been of particular interest to me. I should like to consider with you some of what I believe are its more important problems. I have definite and personal convictions about them; some may appear completely unreasonable. Perhaps they are prejudices; if they are, they are honest ones from which I gladly would be dissuaded. That there are inadequacies in obstetric anesthesia is generally admitted. All of us, nurses and physicians alike, are trying to correct them. Some will be corrected easily and promptly, others with considerable difficulty and not in these times. Certain professional deficiencies distress me greatly. I hope that my remarks about them will not be taken as an attempt to discredit the ethics and dedication of our professions. To the contrary, it is because I respect these so highly that I believe ethics and dedication should never be used to protect or excuse deficiencies in the practice of our professions.

It is neither my intention nor desire to precipitate a bitter argument or to incite a feud. Rather, I wish to provoke constructive thought among all who have a stake in obstetric anesthesia. I hope also to be able to suggest persuasively how the nurse anesthetist can take a more effective and efficient part in arriving at practical answers to some of our common problems.

It should no longer be charged that obstetric anesthesia suffers from neglect. Physician anesthetists are keenly aware of the hazards of administration of anesthesia to the obstetric patient.[8,9] There is a growing interest in this phase of medical care among hospital administrators and public health officials. While considerable discussion about obstetric anesthesia and its problems has reached print, much more takes place at professional meetings, both at hospital and local society levels as well as national ones such as this. Such interest and concern has come about I suspect—and somewhat ruefully, perhaps, — because finally it has been realized that anesthesia for delivery, like the weather, is to be with us every day. It probably is no longer proper to remark, as did the Connecticut sage, that everybody talks about it but

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nobody does anything about it. Yet I am not alone in observing that too little is being done by too many about this important aspect of anesthesia.[1,2,6,8,9]

How vast are the problems? In the hospitals in this country during each day of 1955 some ten thousand babies were born, a total of more than three and a half million for the year. Some form of anesthesia was given for more than two-thirds of these deliveries.

How competently was the anesthetic administered? No one can answer this question, but from what we know it was far below the level both you and I should like it to be. Your association, in its survey of anesthesia service in 1955, could not obtain adequate information about obstetric anesthesia. It did learn, however, that in many hospitals obstetric anesthesia was not given by members from the department of anesthesia, and, moreover, in comparison with persons giving anesthesia for surgery a larger percentage of those who gave anesthesia for delivery were neither nurses nor doctors.[3]

What about fatalities? Deaths attributed solely to anesthesia are far greater on the obstetrical than the surgical services. This is particularly tragic when we realize that deaths due to anesthesia in obstetrics nearly always are preventable. Critical and impartial study of such deaths has shown that in from sixty to seventy-five percent of them the fatal anesthetic was given by those whose understanding and experience must be considered minimal.[11,12] The Dean of American obstetricians, Nicholson J. Eastman, remarked editorially that such anesthetic deaths are "due to ignorance and mismanagement on the part of inadequately trained anesthetists".[4] The findings of your association's survey suggests why one out of every ten maternal deaths results from the anesthetic given. They also point, I believe, to the reason why some three hundred mothers and a far greater number of babies may die unnecessarily this year.

I do not mean this as any direct censure of anesthesia. Anesthesia has contributed importantly to the amazing contemporary advances in medical science. It has outgrown its original mission, the development of the technical ability of making the patient insensitive to pain even though it still is uncertain how that is accomplished. I have great respect and admiration for the anesthetist. I believe that he is much more than a clinical physiologist and pharmacologist practicing sub-lethal toxicology as some have described him.

Modern surgery owes much to anesthesia. While some may grumble about this hand-in-hand relationship they should remember that in its development anesthesia was one of the surgical specialties.[2] Obstetrics also has benefited greatly from research and knowledge in anesthesia. Much has been a sort of hand-me-down from surgical anesthesia. In too many institutions obstetric anesthesia remains the dowdy, unwanted relative who for propriety's sake is remembered and made much of when the occasion demands. This attitude is not exclusive with obstetricians. I quote what anesthesiologists themselves say.[1]

> "While it should be readily admitted that many of the improvements in anesthetic practice have been applied to obstetrics, the contributions in this field have not been commensurate with the progress made in anesthesiology. This is especially true in regard to the administrator, a factor that, in the

final analysis, is the most important to be considered. Most of the interest in obstetric anesthesia has been focused on refinements in techniques and producing better agents, but relatively little has been written or done to encourage the application of these methods with the best judgment and greatest skill".

What is required to be truly qualified in obstetric anesthesia? First it is essential to be experienced and well-trained in the administration of anesthetic agents to patients properly prepared for elective surgery. Then considerable experience in giving anesthesia for vaginal delivery is necessary. Additional training in obstetrics, perhaps as a postgraduate course, should be had since the changed body mechanics and altered physiology of the patient in labor places distinct limitations upon the choice and method of administering anesthetic agents. The problems and hazards created by obstetric complications such as premature labor, toxemia and hemorrhage must be thoroughly understood because these often are serious trials to the ability of the anesthetist. Remember too that obstetric anesthesia requires unusual alertness and competence because delivery always is an emergency where there is little or no time to prepare the patient and that two individuals, extremely important to their community, are liable in widely different ways to the effects of the anesthetic.

Such are some of the challenges inherent in obstetric anesthesia. It is distressing that there are so few experienced and well-trained in it. Why should this be? This is an answer given by anesthesiologists.[1]

"Some anesthesiologists in private practice, while admitting the importance of obstetric anesthesia, avoid as much as they can this important responsibility. They justify this attitude by pointing out that because of its emergency and unpredictable nature obstetric anesthesia encroaches unduly upon the time of the anesthesiologist and interferes with his surgical anesthesia practice. It is argued that it is physically unfeasible to give complete anesthesia coverage to the obstetric department and still run an active surgical schedule. A further pertinent reason given for the depreciation of the importance of obstetric anesthesia is that the financial remunerations frequently are not commensurate with the service rendered".

It is put this way in an editorial in the Canadian Anaesthetists' Society Journal.[5]

"It is unfortunately true that Anaesthesia for the obstetrical patient has received too little attention from anaesthetists. There have been several reasons for this neglect. Many anaesthetists, being otherwise adequately employed, have shunned obstetrical anaesthesia because of its demands upon their time, particularly at night, and because of the numerous rush calls of an emergency nature which it entails. Others have considered it minor anesthesia, taking little real interest in the unique problems involved."

I am deeply disturbed by this reluctance of the physician anesthetist to give proportionately of his time to the administration of obstetric anesthesia, and by his reluctance to accept the responsibility for obstetric anesthesia or at least to assume active supervision of it. To me this suggests a serious deterioration in professional responsibility. I know

that it is silly to expect a physician anesthetist to give every obstetric anesthetic. Nevertheless it is my conviction that as physicians they owe it to themselves, their specialty, to the medical profession at large as well as to the public who supports us to take an active, continuing part in applying available and proper answers to the problems of obstetric anesthesia. I believe that unless the physician anesthetist does this he has little right to the honor and distinction which medicine has earned by years of dedicated professional service. Perhaps this is too severe. Perhaps we are on the threshold of partial specialists in anesthesia. Are we shortly to have neurosurgical anesthesiologists, abdominal surgical anesthesiologists, proctological surgical anesthesiologists, along with obstetrical anesthesiologists? Such would permit a better defense for the limitations of their availability.

Table I
WHO ADMINISTERS ANESTHESIA?

NURSE-ANESTHETIST (A.A.N.A.)	34%
PHYSICIANS (Qualifications unknown)	27%
NURSES (Qualifications unknown)	19%
PHYSICIAN-ANESTHETIST (A.S.A.)	18%
OTHERS (Neither M.D. or R.N.)	2%

Survey of Anesthesia Service 1955—Compton, Bader, Haas, and Lange

To change this strident note and to point to other aspects of the subject I should like to present some factual data. Who administers anesthesia? Table I presents the findings of your association.[3] This survey found that only about half of the anesthetics for surgery were given by persons with known qualifications, 18 percent by physician anesthetists, 34 percent by nurse anesthetists.

Obstetric anesthesia was personally surveyed in 180 hospitals. These hospitals accounted for more than ten percent of the births which occurred in hospitals during the year. They were selected by size and geographic location so that the data might be representative of the country. The findings in Table II do not differ significantly from those of your association. The principal responsibility for obstetric anesthesia showed the expected variations. The percentages for the physician anesthetist and the nurse anesthetist are approximately the same—28.8 percent and 30.5 percent. But note the distribution. Where the service is large, over 3,000 deliveries each year, the physician anesthetist has the principal responsibility. This generally is in the larger hospital. In hospitals having 1,500 to

Table II
PRINCIPAL RESPONSIBILITY FOR OBSTETRIC ANESTHESIA

No. Hosp.	Deliveries per year	M.D. ANEST.	R.N. ANEST.	OBST.	RES or INTERN	OB NURSE
12	Under 500	1	5	1	1	4
15	500-1000	2	6	4	0	3
39	1000-1500	8	7	8	3	13
38	1500-2000	9	15	11	1	2
27	2000-2500	8	9	4	3	3
18	2500-3000	8	4	4	0	2
31	Over 3000	16	9	6	0	0
180	TOTAL	52	55	38	8	27
Percent of total		28.8%	30.5%	21.0%	4.7%	15.0%

Table III

PERSONNEL USUALLY ADMINISTERING ANESTHESIA

No. Hosp.	Deliveries per year	M.D. ANEST.	NURSE ANEST.	** OBST.	RES or INTERN	OB NURSE	Total
12	Under 500	0	2	6	1	4	13
15	500-1000	2	7	9	0	7	25
39	1000-1500	3	16	14	6	12	51
38	1500-2000	3	19	9	5	6	42
27	2000 2500	3	15	7	8	5	38
18	2500-3000	7	8	5	0	3	23
31	Over 3000	8	21	10	4	3	46
180	TOTAL	26	88	60	24	40	238
Percent of Total		11.0%	37.0%	25.2%	10.0%	16.8%	

(**Administers saddle/spinal or pudendal/local)

2,000 deliveries each year the nurse anesthetist is given the principal responsibilty for obstetric anesthesia.

The person given the responsibility for anesthesia on the obstetrical service does not necessarily administer the anesthetic. (Table III). Note that in only eleven percent of these same hospitals did the physician anesthetist usually give the anesthesia—which is less than half of the hospitals where he was considered responsible. In contrast the nurse anesthetist usually gave the anesthetic in 37 percent of these hospitals while she was responsible for the service in only 30.5 percent. Of course, others provide anesthesia service for delivery. The obstetrician along with his intern or resident equals that of the nurse anesthetist; their service is three times that of the physician anesthetist.

Table IV

CRITTENTON

GENERAL HOSPITAL

Detroit Michigan

ANESTHESIA SERVICE 1955	
Surgical	3,398
Obstetrical	2,216
Total	5,614
OBSTETRICAL SERVICE 1955	
Total Deliveries	2,265
Cesarean Sections	95
Operative Vaginal	1,357 (60%)

Table V

OBSTETRIC ANESTHESIA 1955

TYPE	NUMBER	% OF TOTAL
Spinal/Saddle	1,776	80.1
Caudal	90	4.0
Local/Pudendal	30	1.4
General	320	14.5
None	49	
TOTAL	2,265	100.0

If I have observed correctly, most of you work in moderate sized hospitals. I should now like to present figures typical of one of these. Table IV shows that there is the usual three to two relation of surgical and obstetrical anesthetics, 3,398 to 2,216. The number of deliveries, 2,265, approximates the national mean. The various types of anesthesia used are shown in Table V and need no particular comment. The Chief of Anesthesiology is responsible for obstetric anesthesia. He not only arranges the schedule of persons from his department but supervises them as well. He personally trains the residents in obstetrics. Once each month there is a combined obstetric anesthesia con-

ference at which problem cases, complications as well as fetal and neonatal deaths are discussed. These are features I shall mention later.

Typical of this section of the country there is a predominance of conduction anesthesia. Thus it is not surprising to find that the obstetrical resident or the obstetrician gives the greatest percentage of anesthetics for delivery, 85.3 percent, nor that the physician anesthetist gives the least, 4.2 percent. (Table VI) These latter usually are problem cases. I regret that I cannot show the number of cases in which he acted as the anesthesia consultant. I can assure you that it is amazingly high because he is freely available. None of the members of the obstetrical staff were able to recall any complicated or unusual case in which he did not have a part.

Coverage at night, over weekends and on holidays has not been solved. This is a continuing problem involving the variables of schedules, personal objections and preferences and, realistically enough, the dollar-and-cents matters of preferential compensation. In Table VII note the status of the nurse anesthetist. She administered about three out of every four general anesthetics used for delivery, a total of 233. She does not administer conduction anesthesia but in almost a third of them she kept critical watch over the patient who received it. She was an active member of the obstetrical team in almost half of the deliveries for the year. Perhaps because spinal gives more prompt effectiveness she seldom scurries down the corridor with the ether can and cone. Nevertheless she is versatile and competent, as testified by the various agents she used for general anesthesia listed in Table VIII.

In the past the nurse anesthetist was a permanent rather than an occasional member of the obstetric team and contributed importantly to

Table VI

OBSTETRIC ANESTHESIA 1955

TYPE	NUMBER	ADMINISTERED BY			
		M.D. ANEST.	%	OBST or OB Res.	%
Spinal/Saddle	1,776	54	3.0	1,722	97.0
Caudal	90	4	4.0	86	96.0
Local/Pudendal	30	0	-	30	100.0
General	320	36	11.0	51	16.0
TOTAL	2,216	94	4.2	1,889	85.3

Table VII

OBSTETRIC ANESTHESIA 1955

Type	Total	NURSE-ANESTHETIST Given by	%	Assisted	%
Spinal/Saddle	1,776	0		688	38.7
Caudal	90	0		24	26.6
Local/Pudendal	30	0		0	
General	320	233	73.0	0	
TOTAL	2,216	233	10.5	712	32.1

its stability and efficiency. For more reasons than these, obstetricians have loudly deplored her continuing disappearance from the delivery room. During the wee hours of the long nights we spent with her caring for patients in labor we gained deep insight into many of her qualities and abilities. We respected her as a nurse who always showed the skilled observation, the careful attention to detail

Table VIII

CRITTENTON GENERAL HOSPITAL

OBSTETRIC ANESTHESIA 1955

Agents Used For General Anesthesia

N_2O-O_2-Ether	194
N_2O-O_2-Ether-Curare	33
Ether	47
N_2O-O_2-Na Pent-Curare	28
Vinethene	4
Vinethene-Ether	3
Total	320

and the thorough discipline which she acquired from her professional schooling. These, we realized, were the answer to the ease with which she gained her competence as an anesthetist. We loved her for her devotion to duty and for her keen, sympathetic understanding of the laboring mother. Perhaps these qualities endeared her too deeply so that we married too many of them. It may be that we, rather than the lure of the surgeon, caused the current shortage.

These unique qualities of the nurse, we found, were developed further in her schools of anesthesia. In the delivery room she quickly gained a sound and detailed understanding of the vagaries of anesthesia in obstetrics. Guided by the obstetricians with whom she worked and learning by her experience with them, she rapidly developed such proficiency that seldom was there any discussion about the anesthetic she gave. Her capabilities and limitations were mutually realized and respected. We mourn her loss, not only because she was an able and tried member of our team but because we have not been able to develop a similar confidence in the obstetrically inexperienced and undisciplined physician in resident anesthesia training by whom she is being replaced.

Diverted by the surgeon and dominated by the anesthesiologist, it seems to me that the nurse anesthetist rapidly is losing her importance and individuality. The anesthesiologists are extremely short-sighted, if not also lacking in good manners, in their constant degrading of nurse anesthetists. In any organization every member deserves and should have a sense of accomplishment. They should have a sense of belonging, of being a member of the team. In these days of shortages and keen competition we should always remember that the attitude of the executives and department heads usually determines the attitudes of those who make up the organization.

My hackles still rise when I hear the nurse anesthetist called a technician. Yet I must admit that she has become just this in many institutions. I suspect that this has come about with the current distortion of the fee-for-service concept. Medical practice was built upon the unwritten agreement of payment for services given. I find it hard to understand how this permits collecting fees for anesthesia which is induced and then turned over to a nurse anesthetist. If the technician gives no real service or if the fees are lessened, perhaps this practice is not completely wrong. A counter-current seems to be developing; in the coat rooms of many hospitals the term "ghost anesthesia" seems to be replacing "ghost surgery".

The official refusal of anesthesiologists to have anything at all to do with those who have not had similar training[2] is far more disturbing. They have vigorously attempted to discourage schools training qualified nurses in anesthesia. They flatly refuse to take any part in the training of nurse anesthetists. Why such official stand is taken when by themselves they will not be able to meet the demands for anesthesia services in the foreseeable future is beyond my comprehension. Conditions that can be corrected surgically are increasing and more and more women are demanding anesthesia for delivery. I doubt that surgery can be reduced and reproduction cut back until there are sufficient numbers of physicians with approved training to be present at all operations and deliveries.

How available are qualified anesthetists? According to the compilation of the American Medical Association there are some 3,500 physicians actively practicing anesthesia. (Table IX) Your association pointed out[3]) that if all the physicians being graduated from the medical schools in this country were to specialize in anesthesia it would take two and a half years to supply the present needs for surgical anesthesia alone. Nurse anesthetists, some 8,000, augment anesthesia services, but only to the extent that about half of the patients undergoing surgery have the benefit of anesthesia given by a properly trained and qualified administrator.

Last year your association estimated that if anesthesia for surgery were to be given by qualified anesthetists, some 18,000 would be needed. (Table X) Left to physician anesthetists trained in approved residencies, it appears that by 1980 there might be enough of them in our hospitals to give surgical anesthesia. This forecast assumes, however, that all physician anesthetists currently in practice would continue, that none would resign or die, and that the surgical boardings would be at the 1955 level.

Look at the problem in another way for a moment. Let the anesthesiologists take over surgical anesthesia completely. I am confident that the obstetricians would welcome you back to the team. If all the members of your association could be persuaded to leave their technician posts in surgery and take their places as obstetrical anesthetists the deficit for obstetric anesthesia would be small. With over a hundred schools graduating some 800 nurse anesthetists each year, this deficit would be erased in three or four years.

Obviously there is no solution to the current shortage of qualified anesthetists. If our professions are to meet

Table IX

AVAILABILITY OF

QUALIFIED ANESTHETISTS

Physician Anesthetists A. M. A. 1955

ANES. (Not limited or certified)	907
ANES.* (Limited, not certified)	1,314
ANES. A.B. (Limited & certified)	1,139
TOTAL	3,360

Society Membership

American Society of Anesthesiologists (1955)	4,877
American Association of Nurse Anesthetists (1956)	8,076
TOTAL	12,963

the demands of the public for proper and adequate medical care we must join forces. We must cease carping and criticizing the actions and existence of those in our professions. All of us, nurses, physicians, administrators, public health officials, and even business executives should work together for our and the public's common interest. Franklin D. Murphy addressing the American Society of Anesthesiologists emphasized this necessity several years ago.[13]

Table X

ESTIMATED NEEDS FOR QUALIFIED ANESTHETISTS

A.A.N.A. 1950	16,815
A.A.N.A. 1955 (Surgery Only)	17,963

Ratio surgical operations to obstetrical deliveries in most hospitals is 3:2.

ANESTHETISTS NEEDED FOR 100% COVERAGE

Surgery	17,963
Obstetrics	11,974
Total	29,947

> "It is incumbent on everyone who concerns himself with medicine increasingly to orient his actions and thinking towards programs designed to deliver to the American people not only adequate amounts of medical care at a price which people can pay but, perhaps above all else, medical care of the very highest quality."

Meanwhile, and in conclusion, I have a few brief suggestions for the improvement of obstetric anesthesia. Good obstetric anesthesia will result if physician anesthetists will assume the responsibility for obstetrical anesthesia, if rigid rules for the administration of anesthetics in the delivery room by those not specifically trained in anesthesia are established, and if conference type study of the problems of obstetric anesthesia is continuously made in every hospital.

The hospital administrator should: 1) Assure that all the necessary anesthetic and resucitative equipment as well as adequate supplies of drugs and other therapeutic agents are constantly available. 2) Make available an adequate staff. 3) Obtain a physician anesthetist experienced in obstetric anesthesia to actively supervise the service. If none is present in the hospital, arrangements should be made to obtain his availability for consultation and for regular visits to the hospital. 4) Maintain scheduled joint conferences between obstetricians, pediatricians, anesthetists and the administrator. 5) Obtain from the Board of Trustees an agreement that fees charged for obstetric anesthesia will be used only to improve and maintain that service. 6) Subsidize the training in obstetric anesthesia for those who indicate the desire.

The medical staff should: 1) Establish regulations regarding the use of analgesia and anesthesia in obstetrics and actually see that they are followed. 2) Hold joint conferences among those concerned each month. These will do much to break down animosities. They will discover problems of which many can be easily solved to the greater safety and benefit of the mother and her infant. 3) Where the anesthesia staff is small an agreement on priority for emergency cases should be reached so that the obstetric patient will not be penalized because her delivery was not scheduled. 4) Establish training programs in obstetric anesthesia to give at least fundamental knowledge to all persons who serve in the department. Obstetricians, residents, and nurse anesthetists should be given instruction in obstetrics and super-

vised experience in the administration of those agents which they may be expected to use. 5) Actively take part in the recruitment of young women as nurses and nurse anesthetists.

Finally, if all of us who care for the mother during labor and delivery remain constantly aware of the miracle of creation and the holiness of birth we shall gain strength through our dedication and joy in our accomplishments. To all of us I say, noblesse oblige—from those to whom much is given, much is expected.

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Preanesthetic Medication

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Preanesthetic sedation has as its purpose the preparation of the patient mentally and physically so as to facilitate the safe and proper conduct of the anesthesia. The medication may range from a small dose of atropine or scopolamine to complete unconsciousness produced before the patient leaves his bed. Preanesthetic sedation is of utmost importance to the patient, to the surgeon and to the anesthetist. And yet, I believe, these drugs are more poorly prescribed than any others in the pharmacopeia. In many cases they are given improperly as to time, amount and expected pharmacological effect. If properly given, the anesthesia and operation are made much easier for the patient, the surgeon and the anesthetist. However, if improperly administered, the anesthetist is given a struggle throughout the procedure, optimum operating conditions cannot be provided the surgeon, and the safety of the patient may be placed in jeopardy.

What can we expect from proper preanesthetic preparation:?

1. For the patient: a) Psychic depression—the amount of preanesthetic medication should be sufficient to prevent fear and apprehension. It should give the patient a feeling of well-being and euphoria; b) Relief of any pain or discomfort the patient may have; c) Some analgesia before painful anesthetic procedures such as local infiltration or nerve blocks; d) The patient should be assured a good night's rest before operation by the use of sedative drugs. So actually these drugs given the night before operation are part of the preanesthetic medication.

2. For the anesthetist: a) Greater cooperation from the patient if the above things have been accomplished; b) General physiological depression with the following effects: (1) Decrease or absence of secretions making the likelihood of respiratory obstruction and laryngospasm much less; (2) Lower metabolic activity permitting the use of smaller amounts of inhalation or intravenous anesthetic agents; and (3) Preventing or decreasing the chance of the distressing and often times fatal, vago-vagal reflex.

3. For the surgeon: If the patient is mentally prepared for his operation, if the anesthetist has a physiologically depressed patient free from bothersome, perhaps dangerous, secretions, the surgeon has every right in most cases to expect optimum operating conditions .

Properly, most preanesthetic medication consists of a drug which in-

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hibits secretions of the respiratory tract in combination with a sedative or narcotic drug. Atropine and scopolamine are the two drugs commonly used to depress the secretory glands of the respiratory system. Atropine and scopolamine are naturally occurring alkaloids of the belladonna plants. They belong to the group of drugs which depress the parasympathetic nervous system. These drugs have two principal actions in the body. The first effect is on the central nervous system but the mechanism of this action is not known. The second action is that of depression of the smooth muscle and secretory glands innervated by postganglionic cholinergic nerves.

1. Central Nervous System: a) Atropine produces stimulation of the medulla and higher cerebral centers. In the usual doses, there is some central vagal stimulation and moderate respiratory stimulation, which is manifested by an increase in rate and occasionally in depth of respiration. This respiratory stimulation probably comes from a direct action on the respiratory center. b) Scopolamine is quite different in its effect on the central nervous system. While atropine stimulates the brain (and only in toxic doses depresses), the primary action of scopolamine is depression of the higher centers. The usual clinical dose causes drowsiness, relaxation, amnesia and sleep. Like atropine, scopolamine is a respiratory stimulant and not a respiratory depressant as believed by some clinicians.

2. Peripheral Actions: One can predict what the peripheral effects of these two drugs will be if he bears in mind that they are parasympathetic depressants. In a broad sense, the results seem to be those brought about by *stimulation* of the sympathetic system, because with depression of the parasympathetic nerves, the sympathetic activities are unopposed and unbalanced. Atropine and scopolamine differ somewhat in their peripheral action. Scopolamine is much more effective in preventing secretions of the respiratory tract while atropine produces a greater and more prolonged relaxation of the smooth muscle of the bronchi and bronchioles.

a) Respiratory Tract: Both atropine and scopolamine inhibit secretions by blocking the parasympathetic nerves to the secretory glands of the nose, pharynx, trachea and bronchi. Scopolamine is much more effective and acts over a much longer period of time than atropine for the drying action on the secretions of the respiratory tract. The smooth muscle of the bronchi and bronchioles is relaxed by both drugs but to a greater degree by atropine.

b) Circulatory System: Scopolamine has little effect on the heart. The main effect of atropine is to alter the cardiac rate. In the usual clinical doses one may see a slight slowing of the heart rate because of a mild central stimulation of the vagal nuclei in the medulla. In large doses, and often in children, there is a depression of the vagal inhibition and an increase in heart rate results. This increase occurs, because with parasympathetic depression, the sympathetic action of increasing the heart rate in then unopposed. In adequate doses atropine is effective in preventing and treating vagal reflexes which produce a marked bradycardia or even complete asystole.

The usual clinical doses of atropine and scopolamine have little or no effect on the circulation. However, toxic doses of atropine will produce

an active vasodilatation of cutaneous blood vessels.

c) Sweat Glands: There is a strong inhibition of the sweat glands by both atropine and scopolamine and the skin becomes dry and hot. This action becomes very important in surgery during the summer months, especially in the younger age group. Adequate measures must be taken to prevent the development of hyperthermia.

The narcotic drug which is the most efficient and the most commonly used is morphine. Demerol has gained some popularity within the past few years. Pantopon and dilaudid offer no particular advantage in preanesthetic medication over morphine and are seldom used. Some surgeons prefer to use codeine for children.

Morphine is a naturally occurring alkaloid and is one of several pharmacologically active constituents of opium. It was first used as a preanesthetic drug by an Italian, Lorenzo Bruno in 1850. It has stood the test of time and is the most efficient preanesthetic drug that we have. Morphine exerts many effects upon the body but let us confine our discussion only to those which interest us as anesthetists. This drug produces three very desirable actions: 1. It produces psychic depression and brings about disappearance of fears and anxiety. A feeling of euphoria and drowsiness follow its injection; 2. Morphine is the best analgesic drug known and any pain or discomfort the patient may have is relieved; 3. It decreases the metabolic activity of the body and so reduces the amount of anesthetic agent required for a comparable level of relaxation. The use of morphine has some disadvantages, also: 1. Respiratory depression. This disadvantage is eliminated if the medication is given at the proper time before the induction of anesthesia. Studies have shown that the maximum respiratory depression comes thirty minutes after subcutaneous injection, seventy five per cent of normal at one hour, and is negligible after ninety minutes. However, the general physiological depression brought about by morphine (and which certainly is desirable) increases progressively and is at a maximum about two hours after subcutaneous injection. Respiratory depression makes for slow and difficult induction with inhalation agents. 2. Nausea and vomiting are seen with some frequency when morphine is given alone, but occur much less when atropine is given along with morphine, and very rarely with a morphine-scopolamine combination. 3. Loss of vasomotor control. One may see vertigo, hypotension and syncope if patient assumes an erect position after large doses of morphine. But certainly the patient should not be subjected to any activity after receiving his preanesthetic medication.

4. Postoperatively, large and frequent doses of morphine will depress the cough reflex and one may see an increase in pulmonary complication. However, tracheobronchial toilet, smaller doses of analgesic drugs and early ambulation should decrease or eliminate such complications.

Demerol or meperidine is a synthetic spasmolytic and analgesic agent. It has both atropine-like and morphine-like properties. One probably sees less nausea and vomiting with Demerol than with morphine. It produces less depression of the cough reflex. There is less respiratory depression following an injection of Demerol but it is not as free from this effect as the manufacturers would have us believe. Demerol has several real disadvantages in so far as preanesthetic medication is concerned.

This drug produces little or no psychic depression. Nor does it lower general metabolic activity as well as morphine. It is much less effective for pain relief than morphine, standing about midway between codeine and morphine in its effectiveness as an analgesic agent. And finally Demerol is several times more expensive than morphine.

Codeine is another of the opium alkaloid family and is the least potent of the group. It has little value as preanesthetic medication. Codeine is only one-fourth as potent as morphine for psychic and general metabolic depression. It has less than one-sixth the analgesic power of morphine. In large doses, codeine may produce restlessness and excitement.

Barbiturates are synthetic compounds which serve as central nervous system depressants and are widely used in clinical practice as hypnotic agents. The short-acting compounds have a definite important place in preanesthetic sedation. Barbiturates should be used in nearly all cases to insure a good night's rest before operation. They also are of value in providing a quiet, restful morning when operation is scheduled for later in the day. Barbiturates produce excellent psychic depression and are probably the best drugs available for this purpose. In usual clinical doses the barbiturates only slightly depress respiration. Even this may well be the result of sedation rather than a direct action on the respiratory center. However, these drugs provide no analgesia. They produce amnesia and inhibition of the higher centers and in the presence of pain, without administering an analgesic drug, one more often than not sees excitement or delirium.

Suggestions Concerning Preanesthetic Sedation

1. Select one drug of each type and learn to use it correctly as to amount of drug required, the time of administration and the expected pharmacological effect.

2. The drugs must never be given in a routine manner. Standing orders have no place in proper preanesthetic medication. Each patient must be considered individually since there are so many variables among which are: (a) Age—Infants tolerate depressant drugs poorly. Children, because of their higher metabolic rate, require greater amounts of drugs per pound of body weight than do adults. Old, debilitated or toxic patients are quite susceptible to the depressant actions of alkaloids and barbiturates, Consequently, the dosage must be lowered. (b) Size—Excessively obese patients should be given the amount of drug they would normally receive disregarding the excess weight. (c) Physical condition—Obviously the robust muscular athlete requires considerably more medication than the asthenic individual. (d) Intelligence—The intelligent patient to whom procedures can be made clear,usually requires less premedication than the mentally dull person to whom everything remains mysterious in spite of adequate explanations. (e) Psychic state—The nervous apprehensive individual must receive much larger amounts of sedative drugs to produce the same degree of tranquility and depression obtained in a stolid, phlegmatic patient with less medication. (f) Metabolic activity—The amount of preanesthetic medication required is directly proportional to the metabolic rate. The metabolic activity is on the increase until the twenty first year at which time it levels off. It then slowly decreases

until old age when it drops rapidly. Other factors increasing the metabolic rate, and thus requiring greater premedication are hyperthyrodism, fear, pain, and fever. There is a seven percent increase in the metabolic rate for each degree of fever. Conversely, great care must be taken in premedicating patients with hypothyroidism, long standing disease or malnutrtion because even small doses may produce profound depression. It is much better to err on the side of too little rather than too much premedication for patients in poor condition.

3. Understand fully the action of the drugs and prescribe them accordingly. For example, barbiturates and scopolamine have no analgesic properties and their use for pain relief is definitely contraindicated.

4. The use of divided doses adds safety when large amounts of depressant drugs are to be used before anesthesia. This especially useful in premedicating toxic thyroid patients.

5. Patients in shock absorb drugs very slowly from subcutaneous injection because of the decreased circulating blood volume. Therefore, it is wise to use smaller doses of depressant drugs given slowly by the intravenous route. When given by this method, the maximum effect is seen in twenty minutes.

6. Use combinations of depressant drugs with care because the depression resulting from the combination is often greater than the sums of both.

7. Chronic alcoholics, drug addicts and the younger hypertensive individuals are more resistant to both premedication and anesthetic drugs.

8. Morphine should not be used as a premedicating agent before intraocular surgery because of the possibility of postoperative nausea and vomiting. Morphine should also be avoided in patients with increased intracranial pressure since this drug causes a rise in the cerebrospinal fluid pressure and also is a depressant of the respiratory center.

9. Morphine and atropine or scopolamine should be given in a 25:1 ratio. This proportion utilizes the best characteristics of the two drugs and minimizes their undesirable side effects. There is minimal nausea and vomiting and respiratory depression from the morphine and less uncomfortable dryness of the mouth and pharynx from the atropine or scopolamine.

10. The habit of giving preanesthetic medication "on call" is a practice which, more than any other factor, makes for poor anesthesia. The drugs have not had time to produce the effects for which they were intended. The patient has spent the hours since he was awakened, worrying about his coming operation, is given his medication subcutaneously and immediately taken to the operating room. Here he is greeted by strange odors, noises and equipment peculiar to an operating suite. Naturally, his fear and apprehension are increased, with a prolonged induction and perhaps a marked excitement stage resulting. In addition, the anesthetist is plagued by copius secretions with possible respiratory obstructions and all its dire consequences. Because of the lack of general and psychic depression, a much greater amount of the anesthetic agent is required to produce the needed relaxation. Finally, in short procedures following "on call" premedication, the operation may be completed and the patient returned to bed before the medication produces its sedative effects. When the patient sleeps for several hours, more often than not, the anesthetist is criticized for giving too much anesthesia.

Now, permit me to present a method of premedication I have found to be successful in most cases. A short-acting barbiturate, either nembutal or seconal, is given orally at bedtime on the night preceding operation. A check is made in an hour or so and if the patient is not sleeping, the dose is repeated. On the day of surgery, a subcutaneous injection of morphine and scopolamine in the ratio of 25:1 is given ninety minutes before induction of the anesthesia. Morphine is preferred because it produces psychic depression and decreases the metabolic activity of the body. Scopolamine is chosen over atropine because it is much more effective and acts over a much longer period of time in preventing secretions of the respiratory tract, has little effect on the heart, and depresses the higher centers of the central nervous system. The medication is given one and a half hours before induction because at the time of induction the respiratory depression caused by morphine is negligible while the desirable general physiological depression is at its maximum. However, while the above plan is used in the great majority of our cases, it is changed from time to time. To our patients over sixty-five years of age we give a combination of demerol and atropine or a small dose of morphine with atropine. In the older patient, scopolamine occasionally causes excitement, restlessness and hallucinations rather than depression. And too, the average dose of morphine will sometimes produce profound psychic and respiratory depression in the aged. Another exception is made in premedication for open chest cases. Because of its greater effect in preventing the various harmful reflexes of the vagus nerve, atropine is chosen over scopolamine. Asthmatic individuals receive atropine because of its more effective relaxation of the smooth muscle of the bronchi and bronchioles. If the operation is scheduled for mid-morning or later, a short-acting barbiturate is given by mouth early in the day to allay apprehension during the waiting period. This is followed by the morphine-scopolamine combination ninety minutes before induction. The patient in pain presents a little different problem. Neither barbiturate nor scopolamine are given without an analgesic drug. In the presence of pain these drugs often cause excitement and delirium. Premedication for this group of patients consists of an analgesic drug the night before surgery, repeated throughout the night if necessary, and morphine-scopolamine one and a half hours before induction.

Finally, because morphine and scopolamine are effective for three to four hours, we are not concerned if the operation is delayed for a reasonable period of time. If the delay is great, we can give supplementary medication intravenously in the operating room. Likewise, the intravenous route should be used if an operation scheduled for later in the day is substituted at an earlier time.

In conclusion, permit me to quote J. Ross Mackenzie who wrote in the British Journal of Anesthesia in 1931, "Preliminary medication marks one of the greatest advances of anesthetics in recent years. Apart from the anesthetic agent itself there is no single factor in anesthesia that has afforded the surgical patient more physical and mental benefit and protection than the introduction of the preliminary injection of morphine and atropine or the equivalent."

Notes and Case Reports

Heat Sterilization of Spinal Anesthetic Ampules.

In the past spinal anesthetic ampules have been sterilized by immersion in a colored solution of 1:1000 zephiran chloride. The coloring matter used was methylene blue. The antisepsis produced by this method has always been considered adequate. Zephiran chloride, however, is not a completely innocuous solution and it has been shown that tissue damage can result from prolonged contact with it. It has been suggested, therefore, that serious neurological complications following spinal anesthesia might be due to seepage of the liquid antiseptic solution into the spinal drug ampule. Such ampules have been found to have cracks in their walls which are invisible on inspection and yet are patent enough to let small quantities of solution enter into the ampule. The quantity of solution entering into the ampule may be small enough to produce no visible evidence of its presence and still be sufficient to produce neurologic complications.

For this reason anesthesiologists have recommended that spinal anesthetic drug ampules be sterilized by heat rather than by means of a liquid antiseptic. Following the recommendations of our consultant in Anesthesiology, we adopted a technique of heat sterilizing such ampules. The various ampules used are individualy enclosed in glass test tubes in the base of which a colored cotton pad is placed. The cotton pad serves the purpose of cushioning the ampule within the test tube and the colors facilitate identification of the various drugs. The ampules are autoclaved for 15 minutes at a temperature of 250° F. and stored until used. The drugs are never autoclaved more than once.

A sufficient number of ampules must be sterilized at one time to provide a constant source of supply to meet the demands of the surgical schedule. It was found efficient to estimate the need of the surgical

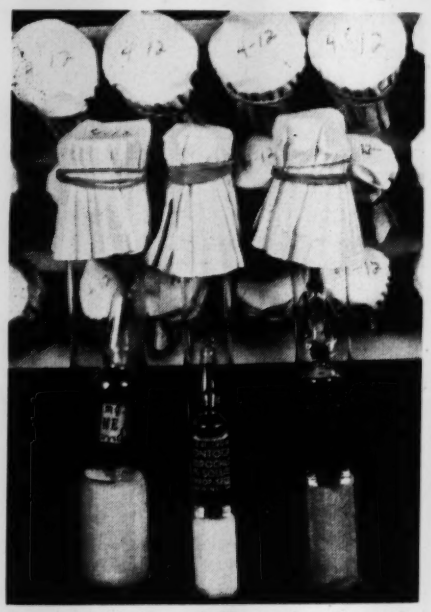

Fig. 1. A photograph illustrating the method of preparing spinal anesthetic ampules for autoclaving. The cotton in the base of the test tubes is colored to assist in identifying the drug.

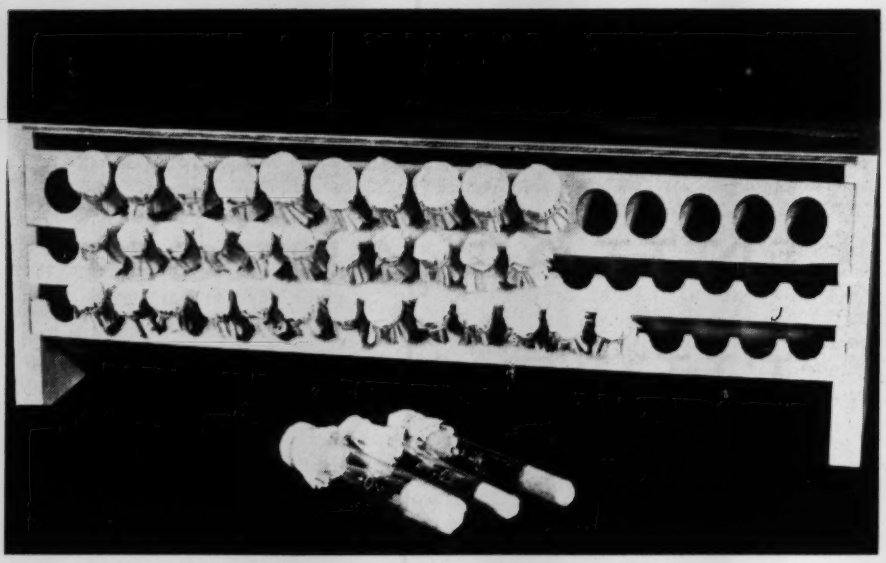

Fig. 2 A photograph of the storage rack for holding the sterilized ampules. It has been designed to fit on the shelf of a storage cabinet.

schedule for a thirty-day period in advance and to sterilize the necessary drugs in sufficient quatities to supply the demand. This resulted in the accumulation of a large number of sterlized ampules which posed a storage and breaking problem. It was found to be desirable to store the ampules in such a way that they would be readily available, easily identified and occupy a minimum of space. To accommodate these requirements, a wooden rack was prepared with slots so arranged that the test tubes containing the spinal anesthetic ampules are safely held and protected from accidental breakage, readily accessible and easily identified. With these modifications in technique we have found it possible to sterilize our spinal drug ampules by heat, as recommended, to have an adequate supply of sterile ampules always available and to store them on shelves with a minimum of wasted space and safe from breakage.

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Legislation

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TAXPAYERS ALLOWED BY COURT TO DEDUCT EXPENSES BETWEEN TWO EMPLOYMENTS "WHILE AWAY FROM HOME"—Section 162 (a) of the Internal Revenue Code of 1954 provides that there shall be allowed as a deduction all the ordinary and necessary expenses paid or incurred during the taxable year in carrying on any trade or business including:

> (2) traveling expenses (including the entire amount expended for meals and lodging) while away from home in pursuit of a trade or business.

A recent case has interpreted the words "while away from home" not to require that the travel must be "away from home overnight" as contended by the Government.

This decision in the case of Chandler v. Commissioner, decided by the United States Court of Appeals for the First Circuit on November 2, 1955, will be of interest to nurse anesthetists who travel between two hospitals involving "widely separarated" areas.

Although, ordinarily, the expenses of transportation from home to work and back are not deductible, the deduction in the Chandler case was allowed by the court. Here the taxpayer, a high school teacher also worked in another city 37 miles away. He claimed a deduction for his automobile expenses in going back and forth twice a week to his place of secondary employment. He resided in the city where his place of primary employment was located.

The deduction was allowed by the court even though the taxpayer worked for two different employers and the travel was not required by the employers and was not for their benefit, except that it permitted the taxpayer to cover both employments. The Court of Appeals held that a distance of 37 miles satisfied the statutory requirement that the travel be "away from home," even though it has been held in a prior case that a trip of 10 miles within the same city as the taxpayer's home was not travel "away from home."

TAX DEDUCTION REGULATION OF BUREAU OF INTERNAL REVENUE FOR REFRESHER COURSES—Nurse Anesthetists may be permitted to take a federal income tax deduction for expenditures in taking short "refresher" courses within their area of practice. The regulation of the Internal Revenue Service setting forth the requirements is as follows:

> 1.162-5. Expenses for education.—(a) (1) In general, a taxpayer's expenditures for his education are personal and are not deductible.
>
> (2) Expenditures for education which are made primarily for the purpose of, or which have the result of, obtaining a position for the taxpayer; qua-

lifying him to enter an employment or otherwise become established in a trade or business or a specialty therein; establishing or enhancing substantially his reputation in his trade or business; substantially advancing him in earning capacity, salary, status, or position; or primarily fulfilling the general cultural aspirations or other personal purposes of the taxpayer are personal expenditures and are not deductible.

(b) As an exception to the general rule in paragraph (a) (1), expenditures by a taxpayer for his education are deductible under section 162 if—

(1) The expenditures are ordinary and necessary for the maintenance of the taxpayer's employment or other trade or business or specialty therein and are directly and immediately related thereto; and

(2) The degree of business necessity and relationship of the expenditures clearly outweighs any personal aspects of the expenditures.

In no event, however, are expenditures which fall under paragraph (a) (2) deductible.

(c) Expenditures by a taxpayer for his education may be deductible under the provisions of paragraph (b) if they are for education of a "refresher" or similar type necessary to maintain (but not to advance) the skills directly and immediately required by the taxpayer his trade or business. Among the factors which will be considered as indicating that education is of a refresher or similar type are that is is especially designed for, and attended primarily by, established practitioners or members of a trade or business for purposes such as keeping abreast of current developments in such trade or business; is of short duration; is not taken on a continuing basis; and does not carry academic credit. Similarly, among factors indicating that the education is not of a refresher or similar type are that it is generally designed for persons preparing to enter an employment or otherwise become established in a trade or business or specialty therein; is of more than short duration; is for the continuing improvement and advancement of the taxpayer's talent or skills; or carries academic credit.

(d) Expenditures made by an employee for his education as a requisite to the continued retention of his salary, status, or employment as a result of an express requirement of his employer may be deductible under the provisions of paragraph (b) whether or not the education is of a refresher or similar type. Thus, expenditures for courses which carry academic credit may be deductible under the provisions of this paragraph. However, expenditures are not deductible under the provisions of this paragraph if, in more than an incidental and relatively minor manner, they have the result of obtaining a different position for the taxpayer; qualifying him to enter an employment or otherwise become established in a trade or business or specialty therein; establishing or enhancing substantially his reputation in his trade or business; or substantially advancing him in earning capacity, salary, status, or position. A taxpayer is considered to have made expenditures as a result of an express requirement by his employer, as a requisite to continued retention of his salary, status, or employment only if the requirement is imposed primarily for a bona fide business purpose of the taxpayer's employer and not primarily for the taxpayer's benefit, and the education has a direct relationship to the duties of the taxpayer's present position.

(e) In general, a taxpayer's expenditures for travel as a form of education shall be considered as primarily personal in nature and therefore not deductible.

(f) Where a taxpayer travels away from home primarily to obtain education the expenses of which are deductible, his expenditures for travel, meals, and lodging while away from home are deductible. However, where as an incident of such trip the taxpayer engages in some personal activity such as sightseeing, social visiting or entertaining, or other recreation, the expenses of such personal activity constitute nondeductible personal or living expenses and will not be allowed as deductions. Where the purposes of a taxpayer's travel away from home are primarily personal, the taxpayer's expenditures for travel, meals, and lodging will be disallowed whether or not the taxpayer participates incidentally in some educational pursuit meeting the requirements of paragraph (c) or (d) of this section. Whether the

purposes of a particular trip are primarily personal or primarily to obtain education meeting the requirements of paragraph (c) or (d) of this section will depend upon all the facts and circumstances of each case. An important factor in making the determination will be the relative amount of time devoted to personal activity as compared with the time devoted to educational pursuits. Expenses in the nature of commuters' fare are not business expenses and are not deductible.

ANNUAL MEETING

A.A.N.A.

September 30—October 3, 1957

Atlantic City, N. J.

The TWENTY-FIFTH QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on May 11, 1957. The deadline for accepting completed applications including the transcripts is April 1. Notice of eligibility will be mailed about April 8.

Applications should be forwarded early enough to allow time to request transcripts and have them returned to the Executive Office before the deadline date.

Insurance

How Much Is Enough?

The recent AANA Convention gave us an opportunity to discuss with many members the Liability Program of the Association.

Of particular interest was the question, "Why can't we get higher limits?" The answer to this question is that higher limits could be made available if enough members requested these limits.

However, a review of the present plan and its progress should first be considered before requesting higher limits against possible lawsuits.

A thorough study of the Liability Plan was made before its final adoption by the AANA. The question of limits on Liability was considered from all points. We knew we had to have a choice of various amounts of Liability. We also realized that a multiple choice of amounts would confuse. Therefore, a minimum and maximum amount had to be selected that would offer sufficient protection at a reasonable cost.

The Insurance Committee, the Legal Department, Executive Department and Insurance Counsellors agreed that too high a limit may induce a member to pay higher premiums for protection that may never be needed.

The Insurance Company did not object to higher limits. They were more concerned with the premium cost on the minimum limit. This is because they, too, made a study of the history of alleged large lawsuits against the Nurse Anesthetists. The Insurance Company will upon request from the Association allow higher limits if the demand for these higher limits warrant such changes.

The progress of the present plan is excellent. We now have over one thousand members enrolled. Many members are only waiting for the expiration date on their present policy to expire before joining the approved AANA plan.

The present plan protecting the members against possible lawsuits arising from either professional or personal acts can be improved to include higher limits, without any additional premium. This depends upon participation and interest by the members in the present plan.

We are hoping by the end of next year to begin negotiations for higher limits without any increase in premium because of the over-all participation in the program. This will, of course, depend on the members' support of the AANA approved program.

In the meantime, we suggest that any member who believes in the need for higher limits write direct to the national headquarters. If there is a demand *now* for higher limits, the AANA will be pleased to entertain the idea of requesting the Insurance Company to make these higher limits available through our present Group Plan.

On Page 309 of this issue there is an explanation of the rates and an enrollment blank for the convenience of the members. Complete this enrollment blank today and mail direct to the national headquarters.

John Maginnis
Insurance Consultant
AANA

AMERICAN ASSOCIATION OF NURSE ANESTHETISTS

PROFESSIONAL AND PERSONAL LIABILITY PROGRAM

An act does not have to be deliberate to be negligent!

As a Nurse Anesthetist, you are legally and financially liable if another is injured as a result of your professional error or omission.

As an individual, you are legally and financially liable if another is injured as a result of personal negligence.

Remembering that "an act does not have to be deliberate to be negligent" you must protect yourself financially, both professionally and personally.

This program offers professional and personal liability insurance in varying amounts at low annual cost.

Plan 1 — $ 5,000/$15,000 Professional Liability plus $10,000 Personal Liability
$9.00 annually

Plan 2 — $10,000/$30,000 Professional Liability plus $10,000 Personal Liability
$12.15 annually

Plan 3 — $15,000/$45,000 Professional Liability plus $10,000 Personal Liability
$13.95 annually

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Book Reviews

PSYCHOLOGY APPLIED TO NURSING. By Lawrence Augustus Averill, Ph.D., Formerly Professor of Psychology, Massachusetts State Teachers College, Worcester, Massachusetts, and Florence C. Kempf, R.N., B.S., A.M., Professor of Nursing Education and Head of the Department of Nursing Education, College of Science and Arts, Michigan State University, East Lansing, Michigan. Cloth. 417 Pages, illustrated. Philadelphia and London: W. B. Saunders Company, 1956. 5th ed.

The authors have given us a book which thoroughly meets the needs of the young student who is entering the nursing field. The graduate will find the book helpful in her approach to and care of the patients who are her responsibility. The chapters on Psychology of the Aging and Aged and the Physically Handicapped are especially applicable in the modern world.

The nurse must not only be able to give adequate care to the aging and aged but she must be well informed on the problems of aging "to aid them to understand their bodies and to transform their resentments and inadequacies into new hopes and achievements within their lessening capacities."

In the United States there are more than five million persons who are suffering from chronic disability of some kind. Science and determination can achieve thrilling ends in rehabilitation. The nurse, by using applied psychology, can help patients to accept and then ignore their deficiencies. It is important for the nurse to aid the patient to reawaken faith in himself and above all in God. Victory is built upon spiritual faith and faith in oneself.

ANESTHESIA FOR OBSTETRICS. LABOR, DELIVERY, INFANT CARE. By Robert A. Hingson, Professor of Anesthesia, Western Reserve University; Director of Anesthesia, University Hospitals of Cleveland, and Louis M. Hellman, Professor of Obstetrics & Gynecology, State University of New York, College of Medicine at New York; Director of Obstetrics & Gynecology, Kings County Hospital. Cloth. 344 pages, 120 figures. Philadelphia: J. B. Lippincott Company. $12.50.

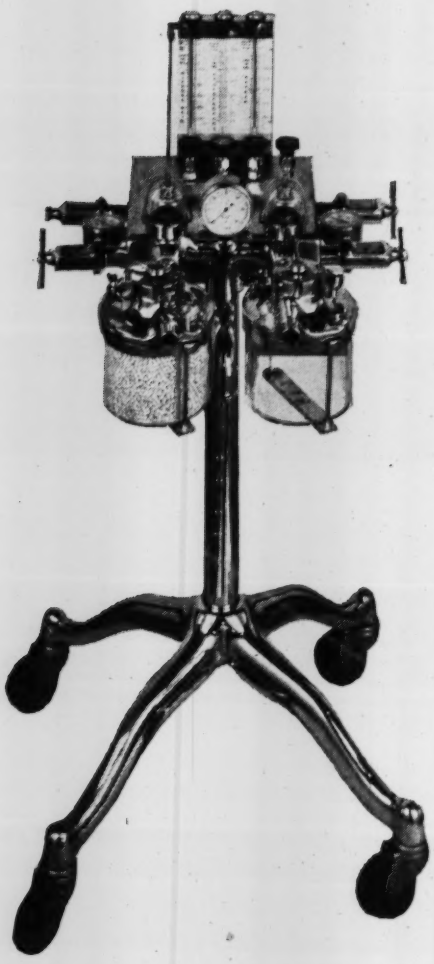
Doctors Hingson and Hillman have presented a very comprehensive study of obstetric anesthesia. They have adequately covered all possible situations that can and do occur in the practice of anesthesia for childbirth. When we think of anesthesia in obstetrics, immediately the many emergencies we have encountered come to mind.

Control of pain in maternal complications such as heart disease, diabetes, toxemias and hemorrhage is very well presented. It is well worth while to refresh one's knowledge of these disease in relation to obstetric anesthesia.

Infant safeguards and resuscitation is given great emphasis. This is as it should be because the ultimate goal is safety for mother and child.

Throughout the book oxygen requirements of the mother and especially the child are stressed.

This book is invaluable for anyone who gives anesthesia for the obstetric patient.



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Classified Advertisements

NURSE ANESTHETIST: for modern air conditioned 5 room operating suite in busy teaching hospital. Work under anesthesiologist. Contact: Assistant Administrator, Mount Sinai Hospital, Minneapolis, Minnesota.

NURSE ANESTHETIST: New 270-bed general hospital with approved school of anesthesia. Salary range $500-$600. Starting salary depends on experience. Scheduled increases. Three weeks paid vacation; 6 holidays per year, sick leave. Classroom teaching optional; additional compensation. Apply: Janet McMahon, R.N., Director, School of Anesthesia, Memorial Hospital, 3200 Noyes Avenue, S.E., Charleston 4, West Virginia.

Openings now available for Nurse Anesthetists. Salary range: Present $4050-$5170; 1/1/57 $4185-$5485. 1000 bed public general, teaching hospital. Write Superintendent, Edward J. Meyer Memorial Hospital, 462 Grider Street, Buffalo 15, New York.

WANTED: Nurse Anesthetists in new 300 bed hospital soon to expand to 600 beds. It is affiliated with The Baylor University College of Medicine. Starting salary $425-$485. On call once weekly, long weekend every 6-7 weeks. Average 42 hour week. Houston is a large metropolitan center for petrochemical industries, boasting a mild climate with resorts, offering numerous recreational advantages. Apply: Director of Anesthesia, Methodist Hospital, Texas Medical Center, Houston, Texas.

WANTED: Registered Nurse Anesthetist for 50 bed new, modern hospital. Pleasant working conditions, good personnel policies. Average number of surgical anesthetics per month, 46. Adequate relief for week-ends and days off. Salary open. Two weeks paid vacation at end of year. Write Administrator, Crawford County Memorial Hospital, Denison, Iowa. State age, training and experience.

NEW YORK STATE: Anesthetist wanted for oral surgery office. Give age and qualifications. Write: Box M-37, Journal of the American Association of Nurse Anesthetists, 116 South Michigan Avenue, Chicago 3, Illinois.

NURSE ANESTHETIST — modern 63 bed hospital located in the Shenandoah Valley of Virginia. Good salary and working conditions. Apply Adminstrator, Stonewall Jackson Hospital, Lexington, Virginia.

NURSE ANESTHETIST: Immediate need for nurse anesthetist for ob. Approved, privately-owned 150-bed general hospital in El Paso. Salary open. For further details reply to Bill Burton, Administrator, Southwestern General Hospital, 2001 Erie Street, El Paso, Texas.

REGISTERED NURSE ANESTHETISTS: Large university hospital using anesthesiologists and nurse anesthetists. Wide variety surgical and anesthetic practice. Salary according to experience. Counting all bonuses new graduates average $400.00 or more per month. Apply: Mrs. Helen M. Geiss, Chief Nurse Anesthetist, Strong Memorial Hospital, Rochester, New York.

Fourth Anesthetist wanted in approved general hospital of 184 beds in city of 25,000. Regulated hours and good working conditions. Four weeks paid vacation; paid sick leave. Salary according to qualifications. Living accommodations in nicely furnished nurses' home. Apply Trinity Hospital, Minot, North Dakota.

POSITION WANTED: Young male A.A.N.A. member desires permanent change to small community hospital or anesthesia teaching school. Prefer last. All agents and technics. Write BoxM-36, A.A.N.A., 116 South Michigan Avenue, Chicago 3, Illinois.

NURSE ANESTHETIST — 90 bed general hospital. 26 new beds under construction, two anesthetist staff, rotating call and week ends off. Apply: Administrator, G. N. Wilcox Memorial Hospital, Lihue, Kauai, Territory of Hawaii.

NURSE ANESTHETIST — 500 bed general hospital doing obstetrical and surgical anesthesia. 250 plus deliveries monthly. $425 - $450 per month — complete maintenance if desired. Apply: Akron General Hospital, Akron, Ohio.

WANTED — Lady Nurse Anesthetist. Group of 7 physicians and 2 nurses. Salary open. Contact Albuquerque Anesthesia Service, Medical Arts Square, N.E., Albuquerque, N. M.

WANTED: Nurse anesthetist for 240 bed general hospital 20 miles from Pittsburgh, Pa. Unusual work day opportunity to work every third day (24 hours at hospital, then 48 hours off) starting September 1. Salary: $4680. All types of surgery except neurosurgery. Apply with references to: S. L. Carpenter, M.D., Chief, Anesthesiology, Allegheny Valley Hospital, Tarentum, Pennsylvania.

NURSE ANESTHETIST: New, modern, 50 bed general hospital; located in town of 6,000; 28 miles from Roanoke, Virginia. Salary open. Apply to: Administrator, Bedford County Hospital, Bedford, Virginia.

NURSE ANESTHETIST: 211 bed general hospital, fully approved. Starting salary $450.00 per month. Ideal working conditions. Apply: James L. Foster, Administrator, Bluefield Sanitarium, Bluefield, West Virginia.

TWO REGISTERED NURSE ANESTHETISTS for 143 bed new and modern, accredited general hospital. Starting salary $450.00 per month plus full maintenance in new modern nurses home, with increase of $25.00 per month after six months. Vacation, sick leave up to thirty days, holidays allowed with pay. Apply to John C. Gilbert, Jr., Administrator of Bristol Memorial Hospital, Bristol, Tenn.

WANTED: NURSE ANESTHETIST. 45 bed hospital. Collect own fees on percentage. Apply: Sister M. Veronica, St. Benedict Hospital, Sterling, Colorado.

NURSE ANESTHETIST: New and modern surgery; unusally strong and well diversified surgical staff. Good opportunity in new 200-bed expanding hospital; college town location; good personnel policies; 40-hour week; 7 holidays, hospitalization, social security. Apply: F. J. O'Brien, Administrator, Chambersburg Hospital Chambersburg, Pennsylvania.

ANESTHETISTS: Charleroi-Monessen Hospital (general, 238 beds and 36 bassinets), North Charleroi, Pa. (HU 3-5561). Salary open plus maintenance, sick leave, holidays, month paid vacation annually, etc. Call or write administrator.

WANTED: Nurse Anesthetist. Position open now. Increasing staff for a 335 bed county general hospital. $400 per month plus meals and laundry. Liberal vacation and sick leave. Pension plans available, AANA preferred. Apply: Medical Director-Adminstrator, Duval Medical Center, Jacksonville, Florida.

ANESTHETIST — Nurse. 600 - bed approved general hospital; excellent salary, one month vacation after a year's service. Apply: Personnel Director, Good Samaritan Hospital, Cincinnati 20, Ohio.

NURSE ANESTHETIST to join obstetrical anesthesia staff. 40 hour week; salary adjusted to experience. Write Administrator, Highland Hospital, Rochester, New York.

NURSE ANESTHETIST: Excellent salary and working conditions. Shore resort community. Send resume of experience and background to Personnel Office, Lawrence and Memorial Associated Hospitals, New London, Conn.

WANTED: Nurse anesthetist for employment in 200 bed accredited hospital in central Pennsylvania. Industrial and farming area. Modern hospital. Department staffed by four nurse anesthetists and certified M.D. Write Administrator, Lewistown Hospital, Lewistown, Pennsylvania.

NURSE ANESTHETIST wanted for 40 bed general hospital. Salary $500 per month. 5½ day week, two weeks paid vacation, sick leave, meals and laundry. Please contact Adminstrator, Memorial Hospital, Dumas, Texas.

NURSE ANESTHETISTS (2): Permanent position in general hospital. Salary dependent on experience. Month's vacation with pay after one year of employment. Two week sick leave, ten paid holidays. Rotating nights; no O.B.S. call. Apply: Alyce J. Benn, Chief Nurse Anesthetist, Massachusetts General Hospital, Fruit Street, Boston 14, Massachusetts.

ANESTHETIST. 400 bed modern hospital. Salary dependent on training and experience. Active surgical and obstetrical services. Call one day a week and every fourth week end. Night call week ends only. Medical anesthesiologist in charge. Social security, low cost life insurance, Blue Cross hospital insurance. Apply: Director of Anesthesia, Methodist Hospital, Peoria, Ill.

NURSE ANESTHETISTS to round out department complement. 150-bed general hospital, 40 minutes from New York City. Pleasant working conditions, good personnel policies, educational opportunities. All types of surgery plus obstetrics. $425 per month plus fee call. Maintenance if desired. Apply: Personnel Director, Middlesex General Hospital, New Brunswick, New Jersey.

REGISTERED NURSE ANESTHETIST. Excellent working conditions in modern 132 bed hospital, Friendly community with two colleges. Salary open pending qualifications and experience. Apply Ralph B. Bersell, Administrator, Passavant Memorial Area Hospital, Jacksonville, Illinois.

WANTED: Nurse Anesthetist, attractive position in 70 bed hospital near Sacramento. Write: Eileen Tipaldo, Yolo General Hospital, Woodland, California.

NURSE ANESTHETIST for 150 bed hospital. Salary open. Contact Administrator, Sacred Heart Hospital, Pensacola, Florida.

NURSE ANESTHETIST. Starting salary $405 per month for members A.A.N.A., $375 per month if eligible for membership. Annual increases plus laundry and private room with bath and telephone in new women's residence. Social Security and Pension Plan. 40 hour week including full time credit for first call. Second call paid for cases done. Six paid holidays, 30 days vacation annually and liberal sick leave policy. Apply: Marshall Kerry, M.D., Chief Anesthesiology, The Reading Hospital, Reading, Pensylvania.

NURSE ANESTHETIST — Permanent night position to supplement staff of six. Salary $415 plus other benefits. Upper midwest location. J.C.A.H. approved hospital. Write Box M-35, A.A.N.A., 116 South Michigan Avenue, Chicago 3, Illinois.

NURSE ANESTHETIST: 195 bed fully approved general hospital. Salary open with annual increments; two in department, three weeks vacation. Write Personnel Director, Blessing Hospital, Quincy, Illinois.

NURSE ANESTHETIST - Oral Surgery Office. Less than 40 hour week. No evening hours, salary open; quiet suburban location. Apply: Drs. Master and Mentel, 511 Millburn Avenue, Short Hills, New Jersey.

NURSE ANESTHETIST to work in municipal hospital. Qualifications: Graduation from a recognized school of nursing supplemented by completion of an accredited course in anesthesia. Possession of a license or eligibility to obtain license as Registered Nurse in the State of Connecticut. Salary range $3692-$4108 per annum. Complete details by writing to Director of Personnel, Municipal Building, Hartford, Connecticut.

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